Politics is for the present, but an equation is for eternity.

- Albert Einstein

University of Alberta

Effects of salmon aquaculture on sea lice transmission and wild salmon population dynamics

by

Martin Krkosek

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of **Doctor of Philosophy**.

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Abstract

Humanity is currently involved in a rapid transition from fishing to aquaculture, which may or may not help restore depleted ocean ecosystems. I combined empirical and theoretical approaches to understand the effect of salmon aquaculture on parasitic sea lice (*Lepeophtheirus salmonis* and *Caligus clemensi*) transmission and wild Pacific salmon population dynamics. Pacific salmon exhibit a characteristic that I call *migratory allopatry* - a period of spatial separation between adult and juvenile hosts that is caused by host migration and that prevents parasite transmission from adult to juvenile hosts. Juvenile salmon enter the ocean uninfected 2-3 months before the return of infected wild adult salmon, keeping *L. salmonis* prevalence $< 5\%$ on juvenile pink salmon (*Oncorhynchus gorbuscha*) in areas without salmon farms. Salmon farms augment host abundance on wild salmon migration routes near spawning rivers, which undermines migratory allopatry and allows lice to spread to wild juvenile salmon. By mathematically combining datasets of *L. salmonis* transmission and pathogenicity on wild juvenile pink and chum (*O. keta*) salmon I find that farm salmon were the primary source of infestations that extended over 80 km of wild salmon migration routes and induced 9-95% mortality in wild juvenile salmon populations. An empirically parameterized model shows high sensitivity of salmon populations to increased *L. salmonis* exposure, predicting population collapse at 1-5 motile *L. salmonis* per juvenile pink salmon despite compensatory predation. These predictions are confirmed by independent analysis of pink salmon abundance data, which show louse infestations have depressed some wild pink salmon populations and placed them on a trajectory toward rapid local

extinction. The louse-induced mortality of pink salmon was commonly over 80% and exceeds previous fishing mortality. If outbreaks continue, then a transition from historical abundance to 99% collapse is expected in four salmon generations. An estimated threshold abundance of 1.3 motile stage *L. salmonis* per juvenile pink salmon divides salmon extinction and recovery. These findings suggest novel parasite transmission dynamics due to salmon aquaculture can erode the capacity of a coastal ecosystem to support wild salmon populations.

for alex

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Chapter 1

Introduction

Ocean fisheries and ecosystems are in decline (Worm et al., 2006). Many coastal seas that were once immensely productive are now depleted (Jackson et al., 2001; Lotze et al., 2006). Fisheries have sequentially targeted species at lower trophic levels (Pauly et al., 1998; Essington et al., 2006) in more remote ecosystems (Myers and Worm, 2003; Pauly et al., 2003). Collapsed stocks show low resilience (Hutchings, 2000) and marine ecosystems have seen phase transitions (Duffy-Anderson et al., 2005) and trophic cascades (Frank et al., 2005; Myers et al., 2007). There are examples of sustainable fisheries (Hilborn et al., 2003*b*; Hilborn, 2007), but fisheries can no longer supply the growing global demand for seafood (FAO, 2007). Nearly half the seafood now consumed by humanity comes from aquaculture, which has expanded rapidly in intensity, geography, and species (FAO, 2007; Duarte et al., 2007). But aquaculture may not realize its potential to augment fish supply, relieve fishing pressure, and enhance sustainability (Naylor et al., 2000). Aquaculture and fisheries are embedded in human economies and ocean ecosystems, and such coupled human-environment systems often contain complex feedbacks and dynamics characterized by thresholds, uncertainty, and surprise (Paine et al., 1998; Folke et al., 2004; Liu et al., 2007). Current market economies favour intensive net pen aquaculture of carnivorous fishes (Naylor et al., 2000), but this may deplete wild fish that are caught to feed farm fish (Goldburg and Naylor, 2005), contribute to eutrophication (Folke et al., 1994), genetically contaminate wild stocks (Fleming et al., 2000), introduce invasive species (Volpe et al., 2001, 2000), and spread infection to wild stocks (Heggberget et al., 1993; Gaughan, 2001; Costello, 2006). In other words, aquaculture may undermine rather than enhance sustainability of ocean fisheries and ecosystems.

Emerging infectious diseases are an example of surprise in human-environment systems because they arise via interactions among humans, wildlife, and domesticated animals (Figure 1.1; Daszak et al., 2000). Human health threats such

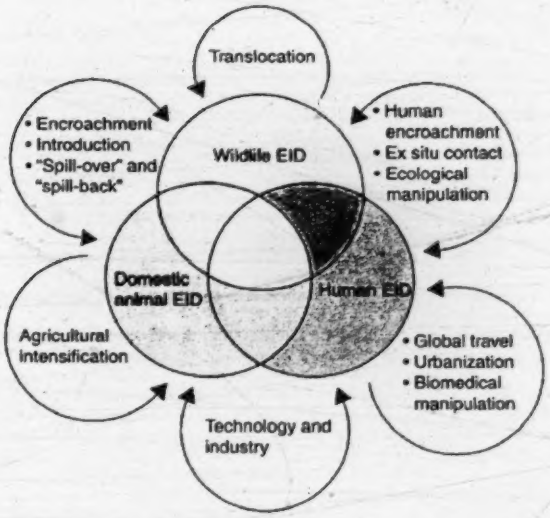

Figure 1.1: The processes underlying many emerging infectious diseases (EIDs) involve interactions among humans, wildlife, and domesticated animals. The figure is from Daszak *et al.* 2000, *Science* 287, 443-449.

as HIV/AIDs (Gao et al., 1999), West Nile virus (Wonham et al., 2004) and H5N1 avian influenza (Kilpatrick et al., 2006) are familiar. Most wildlife examples are terrestrial, where the spread of parasites from livestock has reduced the abundance (Hochachka and Dhondt, 2000) and resilience (Jolles et al., 2005) of some wildlife populations and challenged conservation efforts (McCallum and Dobson, 1995; Hudson et al., 2001). In the oceans, disease outbreaks are increasing (Harvell et al., 2004), in particular for turtles, corals, mammals, urchins, and molluscs (Ward and Lafferty, 2004). The underlying mechanisms are varied and may interact, but include climate warming, pollution, aquaculture, fishing, protection, and transmission from terrestrial hosts (Lafferty et al., 2004). Examples include coral disease increase with pollution and climate warming (Bruno et al., 2003, 2007), urchin population regulation shifting from predation to disease following lobster depletion (Lafferty, 2004), and transmission of phocine distemper virus from canids to seals (Osterhaus and Vedder, 1988). For wild marine fishes, disease outbreaks have declined probably due to fishing (Ward and Lafferty, 2004) because fishing reduces host density which reduces parasite transmission and abundance (Anderson and May, 1978; Arneberg et al., 1998), a process known as "fishing out parasites" (Dobson and May, 1987).

Fishing out parasites does not mean new diseases are not emerging in wild fish, particularly in association with net pen aquaculture. Australian tuna farms that import foreign sardine/pilchard *Sardinops sagax* as feed are the geographic

origin of an introduced herpes virus strain (Gaughan, 2001) that spread through wild *S. sagax* stocks at rates exceeding 10,000 km per year (McCallum et al., 2003). The movement of farm salmon stock among freshwater farms and hatcheries spread the ectoparasite *Gyrodactylus salaris* among Atlantic salmon (*Salmon salar*) populations in at least 30 Norwegian rivers in the 1980's (Heggberget et al., 1993). The import of farm salmon smolts from Scotland to Norway in 1985 began an epidemic of furunculosis, caused by the bacterium *Aeromonas salmonicida*, that swept through the Norwegian west coast, infecting over 500 farms and at least 66 salmon rivers (Johnsen and Jensen, 1994). Transport of farm salmon stock is largely responsible for the spread of an infectious haematopoietic necrosis (IHN) epidemic among salmon farms in Pacific Canada that killed over 12 million farmed Atlantic salmon (*Salmon salar*) (Saksida, 2006). It is not known if the IHN epidemic spread to wild fish stocks, but all Pacific salmon species (*Oncorhynchous* spp.) are susceptible (Roberts and Shepherd, 1997) and migrate through the infected regions (Groot and Margolis, 1991; Saksida, 2006). Salmon lice *Lepeophtheirus salmonis* have infested farm salmon and sympatric wild juvenile salmonids in Europe and Pacific Canada (Costello, 2006). These examples involve pathogens that ultimately originated in wild fish but gained new transmission modes via aquaculture practices, a common mechanism underlying disease emergence (Daszak et al., 2000).

Diseases have emerged spectacularly in farm fishes, concurrent with the rapid growth of the fish farm industry (Kent, 2000; Murray and Peeler, 2005). The causes of disease emergence can be more subtle than humans transporting pathogens or simple transmission from wild stocks, as occurs for IHN (Saksida, 2006) or sea lice (*L. salmonis* and *Caligus* spp.) (Costello, 2006). For example, vertical transmission of avirulent strains of infectious salmon anemia virus (ISAV) may maintain the virus within industry production cycles with disease outbreaks occurring after the virus mutates to more virulent strains (Nylund et al., 2007). Aquaculture has created numerous novel host-parasite associations for salmonids, both within and outside their native ranges (Kent, 2000). Examples are abundant and include Cestode, Copepod, Isopod, Digenean, Microsporidian, Myxozoan, and Sacromastigophoran parasites, some of which are highly pathogenic (Kent, 2000). Aquaculture may even facilitate the evolution of parasitic lifestyles in previously free-living species (Nowak, 2007). *Neoparamoeba* spp. and *Uronema* spp. are free living protistans with direct life cycles that are now found to infect many species of farm fish but have never been reported for wild fish even though disease symptoms are conspicuous (Nowak, 2007). There are myriad ways aquaculture can lead to disease emergence and this will continue to challenge the industry as it grows (Murray and Peeler, 2005).

The rise of emerging disease in farm-wild fish systems may affect wild fish

populations and cascade through marine ecosystems and human societies. The herpes epizootics of *S. sagax* are the largest mass mortalities recorded for a wild marine species (Ward et al., 2001*b*; Gaughan, 2001). The epidemics resulted in range expansion of competing fish (Ward et al., 2001*a*), dietary shifts in seabirds (Bunce and Norman, 2000), reproductive failure and increased mortality in penguins (Dann et al., 2000), and impacts on the commercial fishery and related economic sectors (Gaughan et al., 2000). Wild Atlantic salmon are prized sport fish that experienced increased mortality during the Norwegian furunculosis epidemic and have been extirpated from over 30 Norwegian rivers by the *Gyrodactylus* epidemic (Heggberget et al., 1993). Salmon lice are pathogenic to juvenile salmonids and are a conservation concern for Atlantic salmon as well as sea trout *Salmo trutta* (another prized sport fish) in northern Europe (Costello, 2006). Canadian Pacific salmon - which support coastal ecosystems (Schindler et al., 2003), large commercial fisheries (Groot and Margolis, 1991), and multi billion dollar wilderness tourism industry (www.wilderness-tourism.bc.ca/) - may also be threatened by recurrent salmon lice infestations associated with salmon farms (Morton et al., 2004, 2008). Although there are examples of emerging disease associated with mortality of individual fish (Morton and Routledge, 2005) and isolated instances of wild fish mass mortality (Ward et al., 2001*b*), the evidence for sustained population depression of wild fish (aside from *Gyrodactylus* extirpations of Atlantic salmon) from aquaculture related disease is weak and has been contentious for decades (McVicar, 1997; Noakes et al., 2000; Olivier, 2002; McVicar, 2004; Hilborn, 2006).

1.1 Emergence of sea lice infestations

The most persistent, damaging, and widely distributed emerging pathogens in wild-farm fish systems are sea lice (Johnson et al., 2004; Heuch et al., 2005; Boxaspen, 2006; Costello, 2006). Sea lice are globally distributed caligid copepod ectoparasites of wild and farm fish (Rohde, 2005). They have direct lifecycles with free-living and non-feeding noninfectious nauplii and infectious copepodid stages followed by parasitic copepodid, chalimus, and motile stages (Figures 1.2-1.3) (Costello, 2006). For *L. salmonis*, there are nauplii I and II substages, chalimus I-IV substages, pre-adult I and II and adult motile substages (Johnson and Albright, 1991a). Lice feed on host surface tissues, including skin, muscle, and blood, which can cause lesions, stress, osmotic failure, viral and bacterial infections, and ultimately death (Pike and Wadsworth, 2000; Johnson et al., 2004). Wild fish are the natural reservoir for sea lice that infect farm fish where the parasite can proliferate, costing the aquaculture industry over US$100 million annually in direct mortality, chemical treatment, and lost productivity (Johnson et al., 2004). Caligid infestations of farm fish species

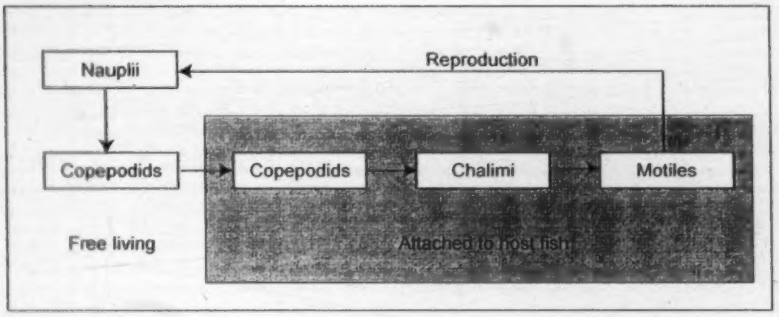

Figure 1.2: The sea louse lifecycle. Free-swimming noninfectious nauplii hatch from gravid motile female lice and then moult into copepodids which infect a host fish or die. Once attached to a host fish, copepodids develop through chalimus stages and then ultimately motile stages which include sexually mature adults, completing the lifecycle. For *L. salmonis*, there are nauplii I and II substages, chalimus I-IV substages, pre-adult I and II and adult motile substages (Johnson and Albright, 1991a)

such as halibut, cod, turbot, and haddock that have recently gained commercial scale production suffer the most damage within the aquaculture industry because control measures are underdeveloped (Johnson et al., 2004). The Atlantic salmon aquaculture industry has long been challenged by *Caligus* spp. and *L. salmonis* infestations in the northern hemisphere and by *Caligus* spp. infestations in the southern hemisphere (Johnson et al., 2004; Costello, 2006). Salmon aquaculture has a long history of research and development on sea lice control that typically involves co-ordinated regional management of production cycles and chemical treatment (Pike and Wadsworth, 2000; Johnson et al., 2004; Costello, 2006). Likely factors that affect lice outbreaks may include adaptation to domestic environments (Murray and Peeler, 2005), density (Lloyd-Smith et al., 2005), temperature (Stien et al., 2005), salinity (Bricknell et al., 2006), chemical treatment (Revie et al., 2002), and evolution of resistance to chemical therapeutants (Fallang et al., 2004).

Sea lice transmission from farm to wild fish and subsequent impact on wild fish stocks has long been contentious (McVicar, 1997, 2004; Hilborn, 2006; Rosenberg, 2008). Wild salmonids are the only fish species for which there are data to evaluate these interactions. Where there are no salmon farms, louse abundance on wild juvenile salmon is low (Tully et al., 1999; Morton et al., 2004). Infestations of wild juvenile salmonids are spatially associated with farms in Scotland (MacKenzie et al., 1998), Ireland (Tully et al., 1999), Norway (Bjørn and Finstad, 2002), and Canada (Morton et al., 2004). Some argue these correlative observations do not demonstrate farms caused the infestations and point out that many factors affect sea lice and

wild fish population dynamics (McVicar, 2004). Sea lice infestations of wild juvenile salmonids can cause physiological and behavioural changes and can kill individual fish (Pike and Wadsworth, 2000; Boxaspen, 2006). It may be that the threat of infestations to wild salmon populations are reduced because parasites can act in compensatory fashion with other mortality factors like predation and competition (Tompkins and Begon, 1999). That is, there may be limited increase in wild fish mortality because a number of dying infected fish will die anyway via predation or competition. Although there have been substantial gains in understanding sea lice and salmon ecology (Costello, 2006; Boxaspen, 2006), there remains enough scientific uncertainty to have slowed the design and implementation of conservation policy. In this dissertation I make advances in this vein by combining mathematical models with large datasets of sea lice transmission and wild Pacific salmon population dynamics.

1.2 Ecology of Pacific salmon and sea lice

The ecologies of Pacific salmon *Oncorhynchus* spp. are dominated by their vast migration between freshwater habitats where they reproduce and the open ocean where they feed (Figure 1.4) (Groot and Margolis, 1991). This migration brings large quantities of nutrients from the pelagic ecosystem to coastal ecosystems and supports wild juvenile salmonids in freshwater habitats (Schindler et al., 2003), as well as many plants (Wilkinson et al., 2005), predators (Reimchen, 2000; Darimont et al., 2003), and scavengers (Christie and Reimchen, 2005; Hocking and Reimchen, 2006) in both terrestrial and aquatic habitats. Most salmonids spend a year or more in freshwater habitats before they migrate to sea and then spend from one to three years in the ocean before returning to their natal river to spawn (Groot and Margolis, 1991; Quinn, 2004). Juvenile Pacific salmon enter the sea in the spring, but pink *O. gorbuscha* and chum *O. keta* salmon are unique among salmonids in their precocious entry into marine waters. Juvenile pink and chum salmon emerge from gravel and immediately migrate to sea in March-May, at sizes approximately 30 mm fork length, and then occupy near-shore (often intertidal) habitats in mixed schools for several months during their migration from coastal to pelagic ecoystems (Heard, 1991; Salo, 1991). Adult Pacific salmon return to coastal ecosystems from the open ocean beginning in July and August (Groot and Margolis, 1991), but pink salmon spend only one year at sea whereas chum salmon spend 2-4 years at sea (Heard, 1991; Salo, 1991). This migration cycle means that juvenile Pacific salmon are not sympatric with large abundances of wild adult salmon for their first 2-3 months of marine life.

The ecologies of sea lice *C. clemensi* and *L. salmonis*, which infect Pacific salmon

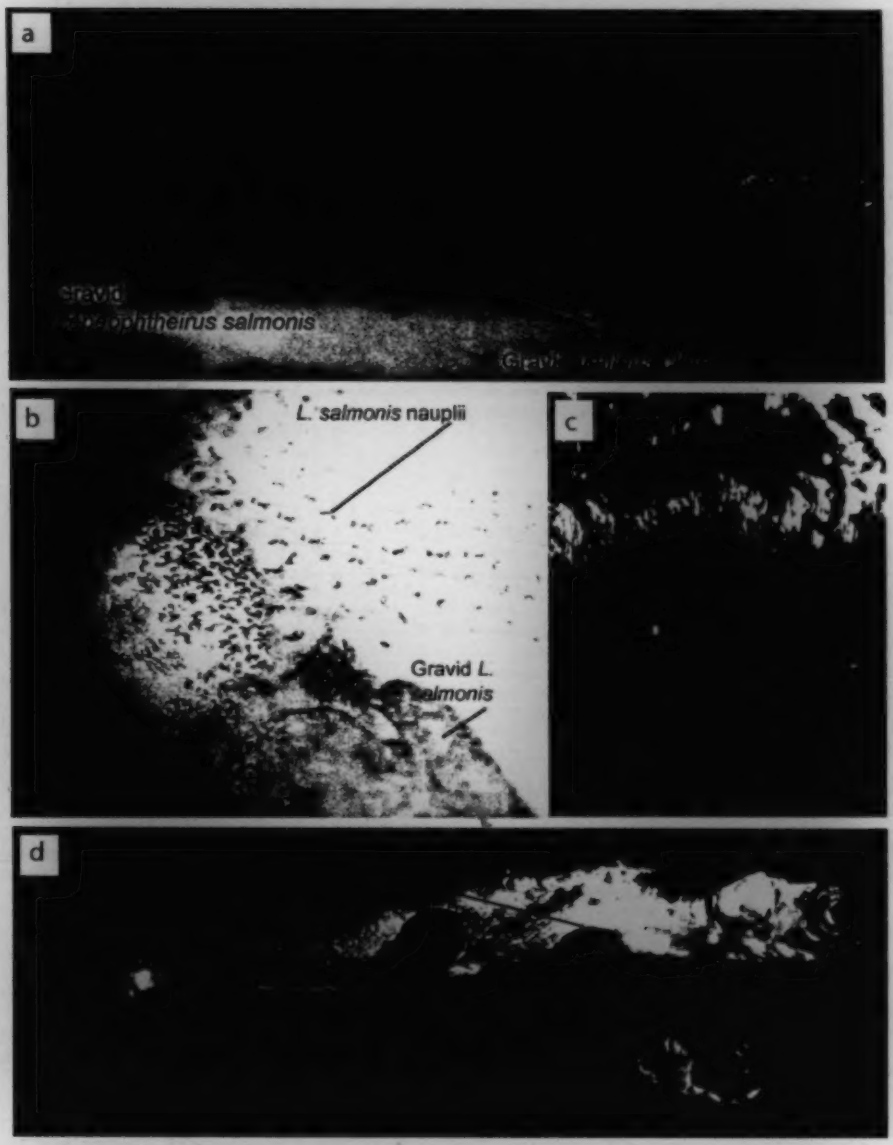

Figure 1.3: Sea lice (*L. salmonis* and *C. clemensi*) on juvenile pink salmon. Shown are gravid female *L. salmonis* and *C. clemensi* on a juvenile pink salmon (a), gravid *L. salmonis* with newly hatched nauplii (b), an *L. salmonis* chalimus larva on a juvenile pink salmon (c), chalimus and adult female *L. salmonis* on a juvenile pink salmon (d). The red colouration in (c) is likely fish blood in the gut of the larval louse. Note the lack of scales and damage to surface tissues scales on the 50-60 mm fork length juvenile pink salmon in (d). All photos contributed by Alexandra Morton.

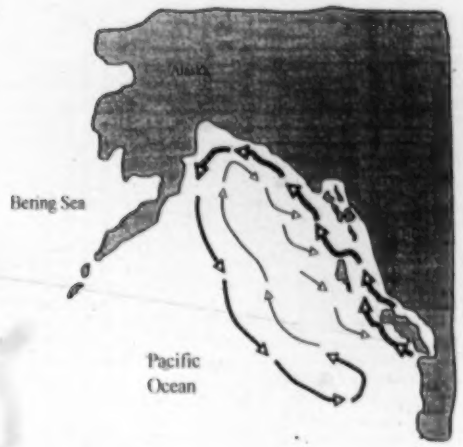

Figure 1.4: The pink salmon migration between coastal and marine pelagic ecosystems. The map was adapted from Heard (1991)

in British Columbia (Figure 1.3 ; Costello, 2006), may be similarly dominated by salmon migrations but also mediated by host diversity. Where there are no salmon farms, salmon lice are rare (prevalence is $< 5\%$) on pink and chum salmon during early marine life probably because most wild adult Pacific salmon that carry the parasite are offshore (Groot and Margolis, 1991; Morton et al., 2004; Peet, 2007). *C. clemensi* has a wider host range than *L. salmonis* (Table 1.1), and the prevalence of *C. clemensi* may be greater on juvenile pink and chum salmon at this time (Morton et al., 2004). One *C. clemensi* infestation of pink salmon fingerlings was reported in 1964 (Parker and Margolis, 1964). The first reported salmon louse infestation of juvenile Pacific salmon occurred on pink and chum salmon in 2001 in British Columbia's Broughton Archipelago (Morton and Williams, 2003), an area with nearly 30 salmon farms. Infestations of juvenile pink and chum salmon in the Broughton have since been recurrent (Morton et al., 2004, 2005). Salmon lice are highly pathogenic to juvenile pink and chum salmon at sizes < 70 mm fork length when the fish have poorly developed scales (Morton and Routledge, 2005). When the fish reach sizes exceeding 100 mm forklength the salmon are more robust to infection (Jones et al., 2006*a*) and it is at approximately this size when they become sympatric with high abundances of returning infected adult Pacific salmon. I call this consequence of host migration on parasite transmission *migratory allopatry* - a period of spatial separation between adult and juvenile hosts, caused by host migration, and which blocks parasite transmission from adult to juvenile hosts. Others have noted it too, suggesting that breaking transmission from adult to juvenile hosts could select for the commonly observed migration between benthic

Host Species	Common Name	Lice spp	Ref
Onchorhynchus spp.	Pacific salmon and trout	L,C	1,2
Salvelinus malma	Dolly varden	L,C	Pers obs.
Salmo salar	Atlantic salmon	L,C	3
Clupea harengus pallasi	Pacific herring	C	1
Gasterosteus aculeatus	Three-spine stickleback	C*	1,4
Hexagrammos spp	Greenling species	C	1
Hydrolagus colliei	Spotted ratfish	C	1
Sebastes spp.	Rockfish species	C	1
Theragra chalcogramma	Alaska pollock	C	1

Table 1.1: Known host species for *L. salmonis* (L) and *C. clemensi* (C). * I do not regard stickleback as a competent host for *L. salmonis* because very few *L. salmonis* survive to adult stages and no gravid *L. salmonis* have been observed on this species (Jones et al. 2006*b*). [1] (Margolis and Kabata, 1988); [2] (Parker and Margolis, 1964); [3] (Johnson and Kent, 1992); [4](Jones et al., 2006*b*).

and pelagic habitats for benthic invertebrates and demersal fish (Strathmann et al., 2002). Salmon farms may threaten wild salmon stocks by undermining migratory allopatry and increasing the exposure of juvenile Pacific salmon to salmon lice.

1.3 Dissertation outline

I will work through four questions to understand sea lice and salmon population dynamics:

(1) How much and how far do lice spread from farms to wild juvenile salmon?

(2) Are juvenile salmon killed by lice from farm salmon and if so how many?

(3) How do natural mechanisms mediate sea lice and salmon population dynamics?

(4) What is the effect of farm-orign lice on wild salmon population dynamics?

I will work through these questions in sequence by combining large datasets with models of sea lice transmission and wild salmon population dynamics. In Chapter 2, I describe, and analyze for measurement error, the field methodology I developed and implemented to count sea lice on wild juvenile salmon without killing the salmon. The methodology allowed me to collect very large datasets of sea lice on wild juvenile salmon in both field and experimental settings. In Chapter 3, I present data and a model of sea lice transmission from an isolated salmon farm to nearby migrating wild juvenile pink and chum salmon. Chapter 4 builds on the sampling design and modeling in chapter 3 by estimating the impact of louse transmission on wild juvenile salmon survival. It combines data on sea lice infections of juvenile pink and chum

salmon migrating past multiple salmon farms with experimental data on the survival of naturally infected wild juvenile salmon collected from the same populations in the same years. In Chapter 5, I investigate the importance of migratory allopatry for pink salmon and sea lice population dynamics. I do so by presenting empirical support for migratory allopatry and then examine a theoretical model that evaluates the sensitivity of salmon population dynamics to increasing louse exposure of juvenile salmon. This chapter also considers the role of host diversity and host migration in sea lice transmission as well as compensatory predation (selective predation on infected hosts) in pink salmon population dynamics. In Chapter 6, I investigate the effect of recurrent salmon louse infestations on wild pink salmon population dynamics by combining five years of infestation data with data on the number of pink salmon returning to rivers on the central coast of British Columbia. All of the fieldwork was conducted in the Broughton Archipelago, east of northern Vancouver Island. In the final chapter, I critically evaluate the results of the chapters and point out directions for future research as well as discus the work in relation to policy.

Chapter 2

Non-destructive enumeration of sea lice on juvenile salmon*

2.1 Introduction

Methods for identifying and counting sea lice on salmon usually involve killing the salmon and storing them for subsequent laboratory analysis (e.g. Jones and Nemec (2004)). The associated demands on human, logistical, and financial resources are extensive owing to the challenges of working in remote field locations. These challenges can limit the scientific scope of research programs because they limit the amount of data that can be collected. In this chapter I describe and evaluate an alternative non-destructive sampling procedure. It reduces resource demands and facilitates the rapid collection of large datasets by analyzing live samples on site rather than dead samples post-season. I analyze the methodology for precision, accuracy, and mortality impacts, and show how the protocol can be extended to inform on fish health. The method is applicable to juvenile pink and chum salmon during their first 1-3 months of marine life and provides a preferable nonlethal alternative to studying depressed or threatened populations.

2.2 Relating morphometrics to condition factor

Fish health is often measured by Fulton's condition factor, which relates the mass of a fish to its fork length by $k = \text{weight} \cdot [\text{fork length}]^{-3} \cdot 100$ (Heincke, 1908; Nash et al., 2006). It is difficult and time consuming to weigh live juvenile salmon but it is possible to use a proxy measure of weight to estimate k based on fork length and body depth measurements. Body depth is the maximum linear distance between ventral and dorsal surfaces, and if this corresponds to the head of the fish, it is

*The methods and results this chapter have been published in Krkošek, M., Morton, M, and Volpe. J.P., 2005. Non-lethal assessment of juvenile pacific salmon for parasitic sea lice infections and fish health. *Transactions of the American Fisheries Society.* 134, 711-716.

measured halfway between the posterior of the head and anterior of the dorsal fin. A simple geometric argument relates these metrics: juvenile salmon morphology is crudely cylindrical or rectangular, and fish weight should be proportional to volume by density. This suggests a power relationship

$$w = \alpha L^{\gamma_1} D^{\gamma_2}, \tag{2.1}$$

where w is weight, L is fork length, and D is body depth. The remaining parameters— α, γ^1 and γ^2—are left to be determined. Taking the natural logarithm of both sides gives

$$log_e(w) = log_e(\alpha) + \gamma_1 log_e(L) + \gamma_2 log_e(D), \tag{2.2}$$

which can be fit to log-transformed fork length-body depth-weight data. The Fulton condition factor then becomes

$$k = (\alpha L^{\gamma_1 - 3} D^{\gamma_2}) \cdot 100. \tag{2.3}$$

2.3 Non-destructive field methods

Juvenile pink and chum salmon were captured with a beach seine off rocky intertidal shorelines (e.g. Jones and Nemec (2004)). The live salmon were subsequently maintained in buckets by periodically exchanging fresh seawater. Still at the location of capture, I examined individual fish by placing them in a clear plastic bag with enough seawater to surround the fish but not enough to cause visual distortion. I used large ziplock storage bags (3.7 L, 27 × 28 cm) with the top 10 cm of the bag removed. I controlled the fish's position and orientation by manipulating the surface tension of the bag and used a hand lens to count and differentiate copepodid, chalimus, and motile lice based on morphology (Kabata, 1972; Johnson and Albright, 1991a). *C. clemensi* were not distinguished from *L. salmonis* for copepodids and chalimi but were for motiles, which have obvious morphological differences (Kabata, 1972; Johnson and Albright, 1991a). It took about 30–90 seconds to examine an individual fish, depending on the fish size and the number of sea lice. The examination included fork length and body depth measurements in addition to louse counts. I measured the body dimensions by laying the salmon, still inside the plastic bag, over laminated graph paper with 1 mm gradation. I also minimized handling time by tasking 1-3 field assistants with the fish examination and another field assistant with data recording. The fish was released at the location of capture following a brief (10-20 minute) recovery period in the buckets.

2.4 Evaluation of the methods

I analyzed 40 juvenile pink and chum salmon using the non-destructive method, and then, within 5 hours, lethally reanalyzed the fish with a dissecting microscope under 8–20× magnification. The identity of each fish was tracked resulting in paired data of live and lethal louse counts and body measurements. I assessed the measurement error by asking: (1) is the nonlethal technique biased to underdetect lice (because it is less thorough)?; (2) are morphometrics equal between the two techniques?; and (3) does sea lice abundance affect the accuracy of the nonlethal technique?

The louse count data were discrete, which violates normality assumptions, and so I applied one-tailed, nonparametric bootstrap paired-sample t-tests to test the null hypotheses that louse counts from live samples were not less than those from lethal samples. Morphometric data did conform to normality assumptions so I applied two-tailed, paired-sample t-tests to test for measurement error in morphometrics. To test the effect of sea lice abundance on the accuracy of the methods I regressed differences between live and lethal counts per fish against the lethal counts on those fish for each louse stage (copepodids, chalimi, and motiles). Statistically significant differences from zero in the y-intercept indicate a fundamental bias between the two techniques while a non-zero slope indicates a bias in the nonlethal technique that is a function of sea lice abundance.

I also evaluated the post-examination short term and long term survival of juvenile salmon. I measured short-term survival by recording the number of mortalities incurred during the analysis of 10,600 (106 sets of 100) juvenile pink and chum salmon in May 2004 in Tribune Channel and Knight Inlet, British Columbia. I measured long-term survival by examining 86 juvenile pink and chum salmon collected on a separate occasion and retaining them for 30 d in a 189-L plastic ocean enclosures at the Salmon Coast Field Station, Simoom Sound, British Columbia. The fish had an average infection burden of 0.24 copepodids, 0.067 chalimi, and 0.077 motiles, and were on average 67 mm fork length. I checked for mortalities and fed the fish commercial salmon feed in excess of satiation every 2–4 h daily. Daily sea surface temperatures were, on average, 12.0°C and ranged from 9.6°C to 14.2°C.

2.5 Results of evaluation

The nonlethal and lethal sampling methods provided similar estimates of louse abundances (Figure 2.1A). However, the nonlethal method was biased to underdetect copepodids ($p = 0.056$) and chalimi ($p = 0.028$), but not motiles ($p = 0.65$; one-tailed nonparametric bootstrap paired sample t-test for each stage). This is reflected in the frequency distributions of measurement error (Figure 2.1B-

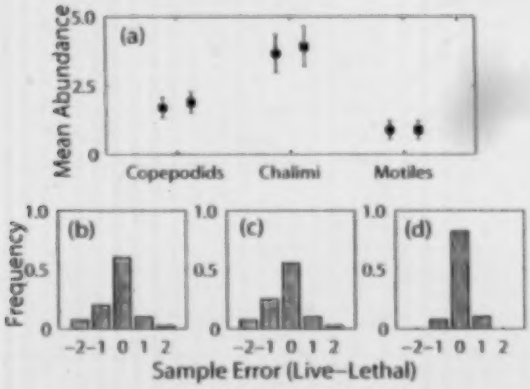

Figure 2.1: A comparison of louse counts between live and lethal sampling methods. Panel **(a)** shows the mean abundances of louse stages estimated by nonlethal (circles) and lethal (squares) methods. Error bars are bootstrapped 95 % confidence intervals on the mean and sample sizes are 40 each. Error frequencies in louse counts are shown for **(b)** copepodids, **(c)** chalimi, and **(d)** motiles, calculated as the difference in counts between paired live-lethal samples.

D); the histograms are negatively skewed for copepodid and chalimus lice, but not for motiles. The mean abundances of lice stages are presented in Figure 2.1, and those estimates ranged zero to four copepodids per fish, 0–10 chalimi per fish, zero to five motiles per fish, and 2–15 total lice per fish.

Regression analyses between differences in live and lethal counts indicated the y-intercept was not different from zero for all lice stages; y-intercepts with 95% confidence bounds were 0.34 (-0.09, 0.77), -0.11 (-0.40, 0.61), 0.09 (-0.20, 0.04) for copepodids, chalimi, and motiles, respectively. The slopes in the regressions were not different from zero for chalimi and motiles (slopes with 95% confidence bounds were 0.09 [-0.20,0.02] and -0.08 [-0.08,0.26]), but the slope was less than zero for copepodids (-0.29 [-0.48, -0.10]; $P < 0.005$). This indicates that as sea lice abundance increase the nonlethal technique will underestimate copepodid abundances, but counts in the other stages will be unaffected.

There were statistically significant differences in morphometrics between live and lethal sampling techniques (length: $P = 3.78 \times 10^{-7}$; body depth: $P = 0.065$; two-tailed paired sample t-test with df = 39 for each). Fork length estimates were greater in nonlethal analyses than lethal analyses, and estimates in body depth from nonlethal analyses were less than those from lethal analyses (Figure 2.2A-B).

We fit equation (3.7) to log-transformed fork length-body depth-weight data from both lethal ($n = 1059$) and nonlethal ($n = 768$) techniques. The log-transformed data showed a strong linear relationship and equation (3.7) explained 95 % and

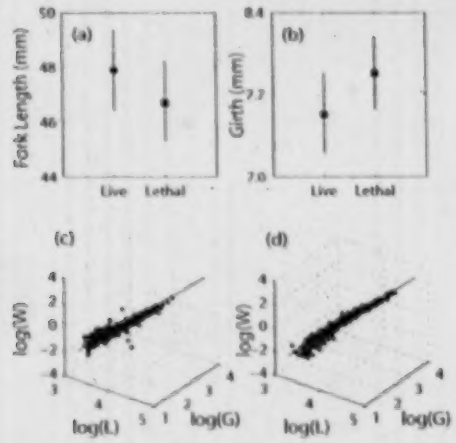

Figure 2.2: Juvenile salmon morphometrics. Top panels: comparison of **(A)** mean fork length and **(B)** body depth between nonlethal (circles) and lethal (squares) sampling techniques. Error bars are bootstrapped 95% confidence intervals on the mean and sample sizes are 40 each. Bottom panels show linear relationships among log-transformed morphometric data for juvenile pink and chum salmons: fork length (L, mm), body depth (D, mm), and weight (W, g). Solid lines are equation (3.7) fit independently to **(C)** live-sampled fish ($n = 736; R^2 = 0.93$) and **(D)** lethally sampled fish ($n = 1059; R^2 = 0.95$). Parameter estimates are given in the main text.

93% of the variance in these data, respectively (Figure 2.2CD). The regressions were strongly significant ($P < 0.001$ for both), and parameter estimates with 95 % confidence limits are: (1) live: $log_e(\alpha) = -9.07$ (-9.36, -8.78); $\gamma_1 = 1.97$ (1.84, 2.09); $\gamma_2 = 0.74$ (0.63, 0.85); and (2) lethal: $log_e(\alpha) = -12.48$ (-13.28, -12.68); $g1 = 3.09$ (2.98, 3.21); $\gamma_2 = 0.21$ (0.18, 0.25).

The average postassay mortality rate was 0.74% per sample. That is, 99.26% of fish subjected to the nonlethal method recovered and were subsequently released. Longterm survivorship was equally good. From 86 pink and chum salmon retained in ocean enclosures following analysis, only one died in the following 30 d.

2.6 Discussion

Both nonlethal and lethal sampling techniques produced similar louse abundance estimates, with a small bias to underdetect copepodid and chalimus lice in nonlethal samples. The measurement error can be attributed to a reduced detectability of small chalimus and copepodid lice, misidentification of louse stages, and reduced integrity of lethal samples. There was a bias to underestimate copepodids as copepodid abundances increased, but there was no corresponding bias in counts of chalimus and motile lice. This bias means that the true abundance of sea lice in

subsequent field and experimental work was likely slightly underestimated.

The differences in morphometrics between lethal and nonlethal sampling techniques were evident from direct measurements and also from differences in parameter estimates of equation (3.7). Differences in body condition between live and dead fish may produce this as live fish retain a firm cylindrical profile while dead fish become flaccid. However, the tight linear relationship among log-transformed fork length-body depth-weight data make it possible to infer weight from fork length and body depth measurements using equation (3.7). Therefore, the same measurements of fish condition (Fulton condition factor) can be obtained from both nonlethal and lethal sampling techniques, although the Fulton condition factor may be uninformative (Morton and Routledge, 2006).

The non-destructive sampling method is only useful during the early marine life of pink and chum salmons when they occupy nearshore habitats and are less than 10 mm fork length. As these fish grow in size they move into deeper waters inaccessible to a beach seine (Groot and Margolis, 1991). For simplicity I grouped both species of salmon and lice together in the present analysis, but this is not a necessary feature of the nonlethal methods since juvenile pink and chum salmon can be easily identified (Pollard et al., 1997). Differences in motile louse counts between the two louse species can provide an index of the presence of these species, if not an index of their relative abundance. If one needs to identify louse species at copepodid and chalimus stages, a lethal subsampling procedure is required.

Overall, the nonlethal technique provides a viable data collection method for analyzing temporal and spatial patterns of louse abundance. However, it likely underestimates the true abundance of sea lice.

Chapter 3

Sea lice transmission from farm to wild juvenile salmon*

3.1 Introduction

Natural host populations can be threatened by pathogens when reservoir host populations are created that maintain or amplify infectious agents (McCallum and Dobson, 1995; Daszak et al., 2000). Where open net pen salmon farms are situated on wild salmon migration routes, there is an opportunity for parasite transmission between wild and farm populations. Although many studies have documented spatial associations of increased sea lice abundance near salmon farms (Costello, 2006), few have worked out the spatial scale and mechanics of sea lice transmission from farm to wild salmon. The transmission process is complicated by the movement or migratory behaviour of wild salmon as well as the dispersion of planktonic non-infectious larval stages of the parasite. In this chapter, I develop a mechanistic spatial model of sea lice transmission and use it to analyze field data of sea lice abundances on juvenile pink and chum salmon migrating past an isolated salmon farm along two narrow and restricted migration corridors in the Broughton Archipelago, British Columbia, Canada.

3.2 Field Methods

I sampled juvenile pink and chum salmon at 1–4 km intervals for 40 and 60 km along two narrow and restricted migration routes relative to an isolated salmon farm (Figure 3.1), and quantified the abundance of copepodid, chalimus and motile stages. An isolated salmon farm (farm A) was situated midway along both migration routes. A second salmon farm (farm B) was situated such that salmon indirectly

*The methods and results in this chapter have been published in Krkošek, M., Lewis, M.A., Volpe, J.P., 2005. Transmission dynamics of parasitic sea lice from farm to wild salmon. *Proceedings of the Royal Society of London Series B.* 272, 689-696.

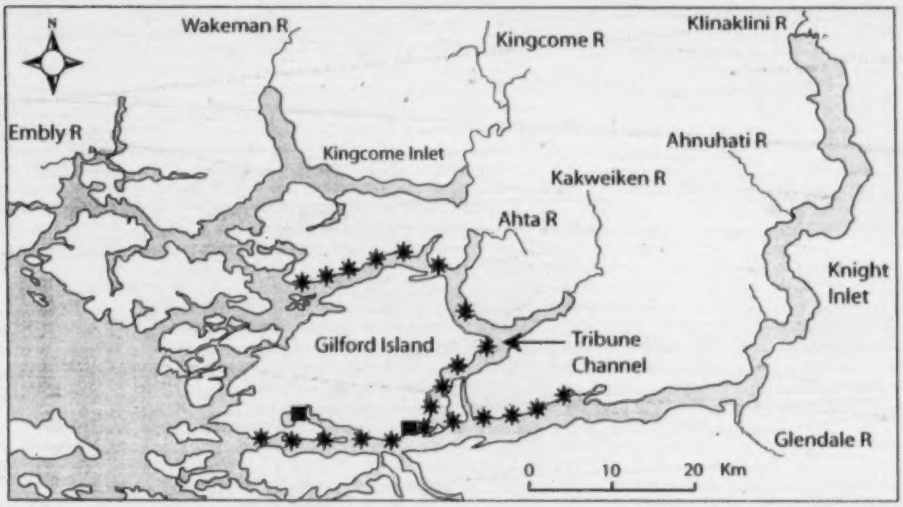

Figure 3.1: Map of the study area showing sample sites (stars) and salmon farms (squares). The separation between the two migration routes corresponding to Knight Inlet (KN) and Tribune Channel (TR) occurs at the location of the salmon near the junction of Knight Inlet an Tribune Channel. The farm at the junction of Knight Inlet and Tribune Channel is Farm A and is the focus of this chapter.

passed within 7 km of it toward the end of the Knight Inlet route. I did not sample for approximately 20–60 km of the migration routes between the landward end of the study area and the various natal streams of the studied populations. There are two replicate sets of samples (79–237 juvenile pink and chum salmon per sample) from each site in the spring of 2003 (17-27 April and 9-23 May). Four datasets result: two replicates of the two migration routes, each representing a spatially structured snapshot of louse population structure. Datasets are labelled: KN-April, KN-May, TR-April, TR-May.

At each site, I collected juvenile pink and chum salmon (measuring 2.8 - 10 cm fork length) by beach seine (30 m long, 4 mm mesh size). The beach seine was drawn in to approximately 1 $m^2 \times 30$ cm and a live subset was retained in 30 L buckets of seawater using a 15×15 cm^2 dipnet. Care was taken to maintain randomness by varying the location, depth and speed of the subsampling procedure. I moved individual fish from a bucket with a $15 \times 15 cm^2$ dipnet and placed them in a 15×27 cm^2 clear plastic envelope for analysis as described in Chapter 2. Temperature readings were taken at most sites and salinity readings were taken at a subset of sample sites around farm A with a Hydrolab Quanta electronic water quality meter.

3.3 Model

The large-scale movement of louse larvae in long and narrow fjordic habitats is limited to movements up and down the habitat length, and I model it with a one-dimensional domain. Juvenile salmon migrate down this domain, initially free of lice, and first encounter infective copepodids that originate from two primary host populations: farm salmon and sympatric wild hosts. As the infection progresses, juvenile salmon become a secondary source of louse larvae themselves. A distinction between the primary sources is their spatial distributions: a farm is a *point source* of lice whereas wild hosts are a *distributed source*. Each source corresponds to distinct spatial profiles in the dynamics of free-swimming and parasitic stages, which form the basis for distinguishing between farm-origin and natural-origin lice in spatially structured data. I formulated three models, corresponding to the possible variations in the origins of sea lice:

- Ψ_0 - includes only natural sources of lice
- Ψ_1 - includes only farm sources of lice
- Ψ_2 - includes both farm and natural sources of lice

3.3.1 Larval distribution models

The study area is modelled in one spatial dimension. The distribution of planktonic copepodids from natural sources is approximated by a uniform spatial distribution: $L_0(x) = \kappa$. First, I derive probability density functions (PDFs) for the spread of nauplii and planktonic copepodids around a point source of arbitrary strength at an arbitrary location, $x = y$. These PDFs then define the spatial distributions of larvae produced by a farm and by lice on the juvenile hosts. The spread of nauplii is modelled by the advection-diffusion equation

$$\frac{\partial n}{\partial t} = D\frac{\partial^2 n}{\partial x^2} - \gamma\frac{\partial n}{\partial x} - \mu_n n \tag{3.1}$$

with the conditions $lim_{x\to\pm\infty} n(x) = 0$. The diffusion coefficient D accounts for the combined effect of tides and random movements of individuals, γ is the advection of larvae due to currents, and individuals die at a per capita rate μ_n. The spatial steady-state solution yields the PDF for the distribution of larvae around the source:

$$k_n(x) = c_n \begin{cases} e^{a_1(x-y)}, & x \le y \\ e^{a_2(x-y)}, & x > y \end{cases} \quad a_2 < 0 < a_1, \tag{3.2}$$

where $a_{1,2} = [\gamma \pm (\gamma^2 + 4\mu_n D)^{0.5}](2D)^{-1}$ and c_n ensures the PDF integrates to 1.

If most planktonic copepodids do not find a host, the spread of copepodids around a point source of arbitrary strength at $x = z$ is given by the same advection-diffusion equation with μ_n replaced by μ_p, the per capita death rate of copepodids. This produces a similar PDF for the distribution of copepodids around $x = z$, given by

$$k_p(x) = c_p \begin{cases} e^{b_1(x-z)}, & x \leq z \\ e^{b_2(x-z)}, & x > z \end{cases} \quad b_2 < 0 < b_1, \tag{3.3}$$

where $b_{1,2} = [\gamma \pm (\gamma^2 + 4\mu_p D)^{0.5}](2D)^{-1}$ and c_p ensures the PDF integrates to 1. In an effort to minimize unidentifiable parameters I fixed $\mu_p = 2\mu_n$, based on longevity experiments (Johnson and Albright, 1991b). The distribution of nauplii around $x = y$ forms a distributed source of copepodids, $k_n(x)$, and the PDF for the resulting distribution of copepodids around $x = y$ is given by the convolution

$$k(x) = \int_{-\infty}^{\infty} k_n(z) k_p(x - z) dz. \tag{3.4}$$

The spread of a copepodids produced by a farm at $x = 0$ is then $L_1(x) = \alpha k(x)$, where α is the abundance of nauplii produced by the salmon farm at the location of the salmon farm. Assuming that the spatial distribution of juvenile salmon is uniform, then M parasitic motile lice per juvenile salmon at location y will produce φ planktonic copepodids, and these copepodids will be distributed according to

$$L_2(x) = \varphi \int_{-\infty}^{\infty} M(y) k(x - y) dy. \tag{3.5}$$

The composite spatial distribution of infective copepodids from all three sources is simply their summation: $L = L_0 + L_1 + L_2$.

3.3.2 Infection model

Infection dynamics on migratory juvenile salmon are linked to these larval distributions by a spatially dependent Poisson process (Papoulis, 1963) that assigns a probability to each datum. In contrast to the common aggregated distributions of macroparasites on their host populations (Shaw and Dobson, 1995; Shaw et al., 1998), the sea lice data conformed well to the characteristics of a Poisson process (Figure 3.2). The mean abundances of copepodid, chalimus and motile stages are

$$C(x) = \beta \frac{1}{\upsilon} \int_{x-\lambda_c}^{x} L(u) du, \tag{3.6}$$

$$H(x) = s_c \beta \frac{1}{\upsilon} \int_{x-\lambda_h}^{x-\lambda_c} L(u) du, \tag{3.7}$$

$$M(x) = s_c s_h \beta \frac{1}{\upsilon} \int_{x-\lambda_m}^{x-\lambda_h} L(u) du, \tag{3.8}$$

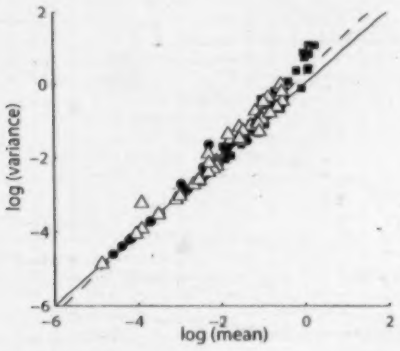

Figure 3.2: Log variance versus log mean for all 41 samples of copepodids (black circles) chalimi (grey squares) and motiles (clear triangles). There are 79-237 fish per sample. The solid line is the variance = mean line which accounts for 96% of the variation. The dashed line is the best-fit linear model, which has slope 1.08 and accounts for 97% of the variation. Compare with fig. 5 in Shaw and Dobson (1995).

where v is the mean seaward migration velocity of salmon, β is the transmission coefficient and s_c and s_h are the proportions of lice surviving copepodid and chalimus stages. The λs are the distances salmon travel in the cumulative mean durations of copepodid (c), chalimus (h) and motile (m) stages. The spatial distribution of infective larvae is $L(x)$: the number of planktonic copepodids within the unit of volume defined by the detection radius of lice centred at x. Only relative values become important, so the detection radius is not explicitly required.

The stochastic infection process assumes salmon encounter planktonic copepodids that attach at a rate β per unit time or $(1/v)\beta$ per unit space. Let τ_c, τ_h and τ_m be the mean durations of copepodid, chalimus and motile stages, respectively, such that $\lambda_c = v\tau_c$, $\lambda_h = v(\tau_c + \tau_h)$ and $\lambda_m = v(\tau_c + \tau_h + \tau_m)$. Let $N_c(x)$, $N_h(x)$ and $N_m(x)$ be spatially explicit discrete random variables for the number of copepodid, chalimus and motile lice on an individual juvenile salmon, respectively. If I assume infection events occur independently then $N_c(x)$ is a Poisson process

$$P\{N_c(x) = k\} = \frac{1}{k!}[I_c(x)]^k e^{-I_c(x)}, \tag{3.9}$$

where I_c is the mean number of attached copepodids at location x, namely $C(x)$:

$$I_c(x) = C(x) = \beta\frac{1}{v}\int_x^{x-\lambda_c} L(u)du. \tag{3.10}$$

The Poisson process has a variable rate parameter (Papoulis, 1963) and spatially explicit mean, $C(x)$. A count of k chalimus lice on an individual salmon could occur from any k of n attached copepodids surviving to the chalimus stage with

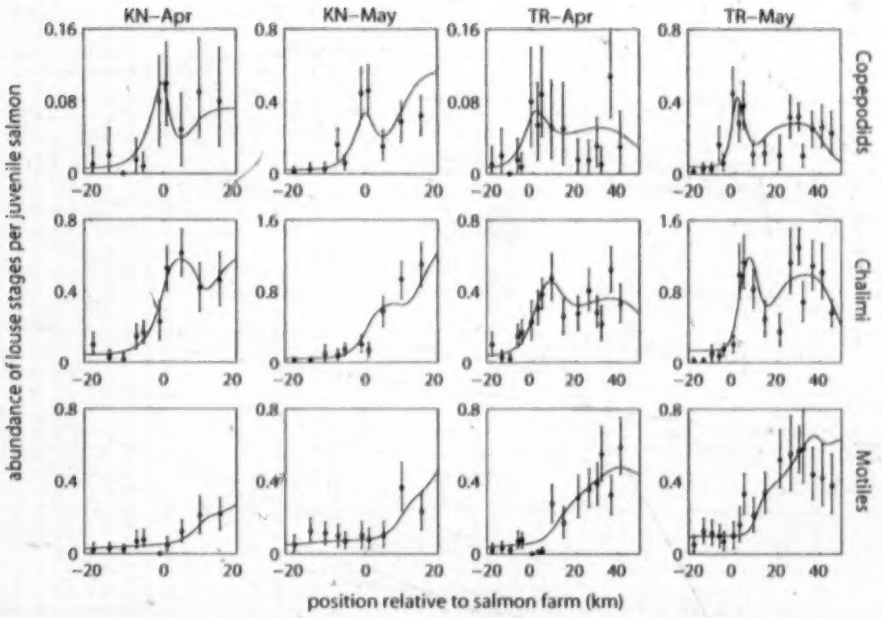

Figure 3.3: Mean abundance of parasitic louse stages on juvenile pink and chum salmon at points along their migration routes relative to a salmon farm located at $x = 0$ (farm A). Salmon migrate in the rightward (seaward) direction. Columns correspond to datasets and rows correspond to louse stages. Error bars are bootstrapped 95% confidence intervals on the means. Solid lines are the $C(x)$, $H(x)$, and $M(x)$ solutions arising from the maximum likelihood best fits of model Ψ_2 to each dataset.

probability s_c. It follows that

$$\begin{aligned} P\{N_h(x) = k\} &= \sum_{n=k}^{\infty} \binom{n}{k} (s_c)^k (1-s_c)^{n-k} \left(\frac{[I_h(x)]^n}{n!} e^{-I_h(x)} \right) \\ &= \frac{1}{k!} [s_c I_h(x)]^k e^{-s_c I_h(x)}, \end{aligned} \tag{3.11}$$

where I_h is the mean number of attached copepodids available for recruitment into the chalimus stage at location x:

$$I_h(x) = \beta \frac{1}{\upsilon} \int_{x-\lambda_h}^{x-\lambda_c} L(u)du. \tag{3.12}$$

Thus, $N_h(x)$ is a Poisson random variable with mean, $H(x)$. In a similar way, I define s_h as the probability a chalimus louse survives to the motile stage and arrive at a Poisson distributed spatially explicit mean for motile stages, $M(x)$

$$\begin{aligned} P\{N_m(x) = k\} &= \sum_{n=k}^{\infty} \binom{n}{k} (s_c s_h)^k (1-s_c s_h)^{n-k} \left(\frac{[I_m(x)]^n}{n!} e^{-I_m(x)} \right) \\ &= \frac{1}{k!} [s_c I_h(x)]^k e^{-s_c I_h(x)}, \end{aligned} \tag{3.13}$$

where I_m is the mean number of attached copepodids available for recruitment into the motile stage at location x:

$$I_m(x) = \beta \frac{1}{\upsilon} \int_{x-\lambda_m}^{x-\lambda_h} L(u)du. \tag{3.14}$$

I assume a time-scale such that only two complete louse life cycles could occur on the juvenile hosts. The solution was found by first solving the larval distribution submodels for the primary sources of lice, $L_0(x)$ and $L_1(x)$, followed by solving equations 3.6-3.8 for the first generation of $C(x)$, $H(x)$, and $M(x)$, followed by solving for the secondary distribution of lice $L_2(x)$ from equation 3.5 to generate $L(x) = L_1(x) + L_2(x) + L_3(x)$, and then finally solving equations 3.6-3.8 to get the solutions for $C(x)$, $H(x)$, and $M(x)$. This process was aided by a fast Fourier transform algorithm in MATLAB when calculating $L_2(x)$. I used maximum likelihood to fit the solutions $C(x)$, $H(x)$, and $M(x)$ to the data using the likelihood function

$$\prod_{s} \prod_{j=c,h,m} \prod_{k_s} P\{N_j = n_k | \Theta\} \tag{3.15}$$

where Θ is the set of parameters to be estimated, s indexes the number of sample sites in a dataset, j indexes the developmental stage (copepodid, chalimus, motile), and k_s indexes the number of fish in sample s. This model fitting process was done separately for each replicate dateset. I used likelihood ratios (Hilborn and

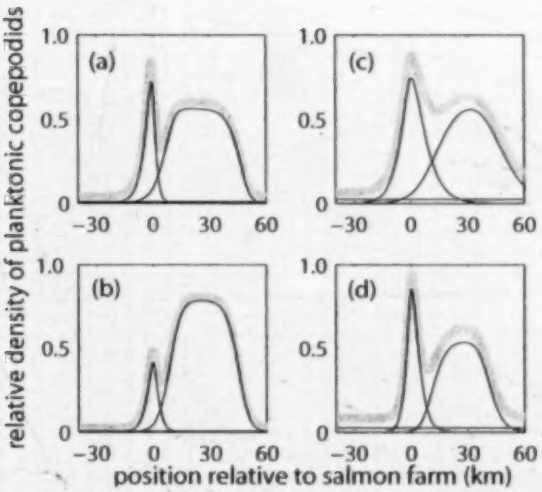

Figure 3.4: The spatial distributions of planktonic copepodids inferred by model Ψ_2 on a relative scale. Juvenile salmon migrate in the rightward (seaward) direction. The thick grey line is the total abundance of copepodids produced by all sources. The horizontal lines near zero are the ambient infection pressures. The thin dark curves oriented about $x = 0$ are the distributions of copepodids produced directly by farm A and the second curves to the right are the distributions of copepodids produced by the farm-origin cohort of lice on the juvenile salmon. The latter distribution was found by solving the model with $\kappa = 0$ to eliminate any contribution of lice from natural sources. Corresponding datasets are I-April (a), I-May (b), II-April (c), and II-May (d).

Dataset	n	df	R	p
I-Apr	3339	1	122.6	< 0.001
I-May	5700	1	51.6	< 0.001
II-Apr	2646	1	57.3	< 0.001
II-May	4857	1	251.0	< 0.001

Table 3.1: Likelihood ratio tests for secondary infection dynamics. (Column n is the number of counts of copepodid, chalimus, and motile lice per fish used in evaluating R, the likelihood ratio statistic. The model used was Ψ_2, and the null model was created by forcing $\varphi = 0$. The degrees of freedom are the differences in the number of parameters between the two nested models.)

Mangel, 1997) to test if the farm did not infect wild juvenile salmon and if secondary infection from the juvenile salmon is significant, and Akaike information criteria (AIC; (Burnham and Anderson, 2002)) to select the best model among Ψ_0, Ψ_1 and Ψ_2. Both species of lice and juvenile salmon were included in the same analysis due to similarities in host behaviour and parasite life cycles. In subsequent chapters I relax this assumption and look at sea lice and juvenile salmon population dynamics at a species specific level.

3.4 Results

A total of 41 samples were collected across the four datasets yielding a total of 5514 juvenile salmon that were sampled for sea lice infections. From these fish, I counted a total of 552 copepodids, 2078 chalimi and 1015 motiles. Of these motiles, there were 12 ovigerous *L. salmonis* females and 53 ovigerous *C. clemensi* females. The distributions of lice on juvenile salmon ranged from slightly underdispersed to slightly overdispersed, and all samples closely followed the $1:1$ variance to mean line, which accounted for 96% of the variability in those data (figure 3.2). Sea temperatures were $8-10°C$ and salinity readings were in the range 27.6–30.3ppt.

The analysis revealed that lice from farm salmon infected wild juvenile salmon (likelihood ratio test, $R > 242.4$, d.f.=8; $p < 0.001$ for all datasets) and that juvenile salmon were a secondary source of infection (likelihood ratio test, $R > 19.8$, d.f.=1, $p < 0.001$ for all datasets). The parameter estimates are shown in Table 3.2. Model selection statistics (AIC) indicated Ψ_2 was the superior model for all datasets; the minimum ΔAIC between all models and Ψ_2 was 22.4 and the probability Ψ_2 was the best model approached unity in each dataset (minimum Akaike weight =0.9998; Table 3.4). These results strongly suggest that both wild and farm primary hosts

were important sources of sea lice infecting juvenile salmon and in addition that juvenile salmon themselves were an important secondary source of sea lice.

The sea lice data were spatially structured: most lice were observed on juvenile salmon after they passed farm A (figure 3.3). Juvenile salmon carried low burdens of lice prior to their encounter with farm A. Near farm A, a large abundance of parasitic copepodids was observed followed by subsequent peaks in copepodids, chalimi and motiles further down the migration routes. The fit of model Ψ_2 agrees well with these data (figure 3.3) and explains these patterns. Prior to passing farm A, louse abundances were at natural ambient levels determined by a balance between immigration and emigration/death rates through each stage. Near farm A, a large cohort of lice colonized the juvenile salmon. These lice developed through subsequent chalimus and motile stages as their hosts migrated, producing the spatially displaced peaks in chalimi and motiles. Larvae were subsequently produced from the motile population on the juvenile hosts and produced the secondary infection waves of copepodids and chalimi apparent in the data.

Due to the mechanistic structure of the model, it was possible to analyse the transmission dynamics across each component in this system using parameter estimates from the maximum likelihood fits of model Ψ_2 to each dataset (figure 3.4). The analysis reveals that larvae originating from the farm and from the farm-produced cohort of lice on the juvenile salmon were responsible for the majority of the infection dynamics observed in the data. These differences can be quantified (table 3.5). Assuming the farm is 0.2 km in length, then the production of infective copepodids by the farm was on average 3.14×10^4 times greater than natural production in this spatial interval. This corresponds to an infection pressure near the farm that was on average 73 times greater than ambient levels and exceeded ambient levels for an average of 30 km. Inclusion of the dynamics of the farm origin cohort of lice on the juvenile salmon suggests that the production of larvae due to the farm was 2.08×10^5 times greater than ambient levels per unit space, with a composite spatial profile of infection pressure that exceeded ambient levels for 75 km (figure 3.4).

3.5 Discussion

The analysis identifies a signal amidst noise in empirical data. The signal is the ensemble of spatial distributions of lice predicted by the models. Any source of error that confounds the model predictions would detract from the results, while any unaccounted sources of variation would add noise to the data and reduce the statistical power of the analysis. There were many unaccounted sources of noise in the sea lice data: variation in temperature and salinity; inter- and intra-specific

Parameter	units	I-Apr	I-May	II-Apr	II-May
$\alpha\beta v^{-1}$	km^{-1}	0.6388	2.1908	1.0108	2.1848
$\kappa\beta v^{-1}$	km^{-1}	0.0001	0.0002	0.0002	0.0008
$\varphi\beta v^{-1}$	km^{-1}	0.0466	0.2457	0.0193	0.0588
D	$km^2 \cdot day^{-1}$	0.2589	0.9505	13.3969	16.1684
γ	$km \cdot day^{-1}$	-0.1	-0.1	0.9578	2.8801
μ_n	day^{-1}	0.1582	0.2182	0.5654	2.8328
s_c	-	0.8804	0.2671	0.7174	0.6510
s_h	-	0.2586	0.3826	0.3805	0.1963
λ_c	km	0.95	0.95	0.85	1.45
λ_h	km	10.35	10.35	10.82	9.50
λ_m	km	49.97	46.54	43.87	39.23

Table 3.2: Maximum likelihood parameter estimates of model Ψ_2. (Parameters: β is the rate at which individual copepoidids attach to a host; v is the average seaward migration velocity of juvenile pink and chum salmon; α is the total abundance of planktonic copepodids produced by the farm; κ is the ambient abundance of planktonic copepodids; φ is the number of planktonic copepodids produced by a motile louse; D is the diffusion coefficient for the spread of nauplii and copepodids; γ is the advection of nauplii and copepodids; μ_n is the per capita maturation rate of nauplii into copepodids; s_c and s_h are the proportion of lice surviving the copepodid and chalimus stages respectively; λ_c, λ_h, and λ_m are the distances a juvenile salmon travels in the cumulative average durations of copepodid, chalimus, and motile stages, respectively.)

Dataset		primary dynamics only			primary and secondary dynamics		
	n	df	R	P	df	R	P
I-Apr	3339	7	190.8	< 0.001	8	242.4	< 0.001
I-May	5700	7	287.4	< 0.001	8	410.0	< 0.001
II-Apr	2646	7	388.8	< 0.001	8	445.2	< 0.001
II-May	4857	7	512.6	< 0.001	8	765.6	< 0.001

Table 3.3: Likelihood ratio tests of the null model: Farms do not infect wild salmon. (Null model, Ψ_0: the farm did not infect juvenile pink and chum salmon. Alternate model, Ψ_2: both farm and natural sources infected juvenile salmon. Column n is the number of counts of copepodid, chalimus, and motile lice per fish used in evaluating R, the likelihood ratio statistic. Ψ_0 is nested within Ψ_2 and the degrees of freedom, df, is the difference in the number of fitted parameters between the two. I present results from models with and without secondary infection dynamics, by forcing φ=0 for the latter.)

variations in lice life-history parameters; inter- and intra-specific variations in juvenile salmon behaviour and host-parasite interactions; mixing of juvenile salmon with different immunological histories; deviations from a mean seaward migration velocity; deviations from a uniform juvenile salmon spatial distribution; temporal variations in the infestation levels on the farm; infection originating from farm B; density-dependent effects on louse survivorship and/or host mortality, potential patchiness in planktonic louse distributions; and generally, the one-dimensional mathematical representation of a dynamic three-dimensional biological system.

The only confounding source of error would occur if a population of natural hosts was aggregated around the farm, producing the spatial distributions I attributed to the salmon farm. Were this to occur, such an aggregated wild population would have to be either four orders of magnitude more dense than anywhere else in the study area or four orders of magnitude more infested than other wild hosts. Furthermore, the spatial distributions in the data require a point source that is stationary for at least two louse life cycles. For *L. salmonis*, this is at least 100 days at 10°C (Johnson and Albright, 1991a,b). Therefore, not only would such a population be unrealistically dense or infested, it would also be unrealistically stationary. Given the paucity of confounding factors and abundant sources of noise, the strength of our results suggests the unaccounted sources of variation must be trivial relative to the effect of the salmon farm. Indeed, the calculations suggest the farm raised infection levels by four orders of magnitude, and it is unlikely that other sources of error could vary by such a magnitude.

The statistical results are strong because those deviations are small relative to the

Dataset	model	$\triangle$ AIC	w_j
I-Apr	Ψ_0	226.4	6.8844×10^{-50}
	Ψ_1	144.4	4.40×10^{-32}
	Ψ_2	0	~ 1
	$\Psi_1(\varphi = 0)$	143.6	6.57×10^{-32}
	$\Psi_2(\varphi = 0)$	49.6	1.70×10^{-11}
I-May	Ψ_0	394.0	2.78×10^{-11}
	Ψ_1	71.0	3.82×10^{-16}
	Ψ_2	0	~ 1
	$\Psi_1(\varphi = 0)$	70.6	4.67×10^{16}
	$\Psi_2(\varphi = 0)$	120.6	6.49×10^{-27}
II-Apr	Ψ_0	429.2	6.32×10^{-94}
	Ψ_1	40.2	1.37×10^{-5}
	Ψ_2	0	0.99998
	$\Psi_1(\varphi = 0)$	22.4	1.87×10^{-9}
	$\Psi_2(\varphi = 0)$	55.2	1.0315×10^{-12}
II-May	Ψ_0	747.6	4.58×10^{-163}
	Ψ_1	168.0	3.31×10^{-37}
	Ψ_2	0	~ 1
	$\Psi_1(\varphi = 0)$	289.0	1.78×10^{-63}
	$\Psi_2(\varphi = 0)$	249.0	8.52×10^{-55}

Table 3.4: Model selection statistics (AIC). (The number of observations used in calculating AIC values is the same as in Table 3.3. Models: Ψ_0 contains only natural sources of infection; Ψ_1 contains only farm-produced infection; Ψ_2 contains both farm and natural sources of infection. Models without secondary infection dynamics are indicated by setting $\varphi = 0$. Setting $\varphi = 0$ did not change the structure of the fitted model Ψ_0 so Ψ_0 is only presented only once for each dataset.)

overall dynamic pattern observed in the data and that pattern could only occur were a salmon farm the primary driver of sea lice dynamics on wild juvenile salmon. This is in agreement with Morton et al. (2004), who found virtually no lice on juvenile pink and chum salmon in several regions of British Columbia without salmon farms. The results are also in agreement with many other European studies finding spatial associations between sea lice and salmon farms (Costello, 2006). Some studies found external oceanographic co-variates more important in determining louse dynamics (e.g. Marshall (2003)), but such studies taken over seasonal time-scales import large temporal variations in temperature and salinity that affect the dynamics of all lice, but tell little about the interactions across wild and farm host populations. I have avoided such confounds by focusing on smaller temporal scales and explicitly examining the interactions among all host populations.

The results in this chapter probably underestimate the true abundance of sea lice due to the minor sampling error identified in Chapter 2. Because the comparisons between farm and wild sources of lice are made on a relative scale this sampling error does not affect the main results on the relative magnitudes and spatial distribution of copepodids originating from wild and farm hosts. The measurement error may have a small effect on the maximum likelihood parameter values, such as overestimating survival from copepodid (underestimated abundance) to motile (accurate estimation). However, this error is very small and nearly symmetric, it will be negligible relative to the noise in the data as well as unaccounted sources of variation such as the mixing of juvenile pink salmon populations from different rivers (such as Glendale, Ahta, and Kakweiken) that have different migration routes and farm exposure.

The data and analysis presented in this chapter extend the current understanding of lice interactions between wild and farm salmon by estimating transmission from farm to wild salmon and then tracking the subsequent dynamics of lice. Initial transmission from farm to wild juvenile salmon may be minor compared with the subsequent spread of the farm-produced lineage of lice. While this chapter tracked the transmission dynamics of sea lice it did not measure the effect on juvenile salmon survival or wild salmon population dynamics. It is possible that sea lice infestations may cause mortality sufficiently high to threaten the wild salmon populations, a possibility that I evaluate in detail in the next chapter.

Statistic	KN-Apr	KN-May	TR-Apr	TR-May	Average
Primary larval production per unit space $\alpha \cdot (0.2\kappa)^{-1}$	3.19×10^4	5.48×10^4	2.52×10^4	1.37×10^4	3.14×10^4
Primary and secondary larval production per unit space $(\alpha + \int L_2(x)d)(0.2\kappa)^{-1}$	1.67×10^5	5.54×10^5	6.95×10^4	4.42×10^4	2.09×10^5
Maximum primary infection pressure $max_x\{\alpha k(x)\}\kappa^{-1}$	87	144	30	32	73
Distance primary infection pressure exceeds ambient levels (km)	25	27	45	23	30
Distance primary and secondary infection pressure exceeds ambient levels (km)	73	76	91	62	75

Table 3.5: Summary of the spatial infection pressures caused by the salmon farm relative to ambient levels. (Larval production per unit space was calculated based on a farm length of 0.2 km. Secondary larval production from the farm origin cohort of lice on the juvenile salmon was calculated by solving the model with $\kappa = 0$ to eliminate any contribution from natural origin lice. Infection pressure is the local abundance of planktonic copepoidids.)

Chapter 4

Sea lice transmission and wild juvenile salmon mortality*

4.1 Introduction

Sea lice infestations of wild juvenile salmon have been commonly observed in spatial association with salmon farms (Costello, 2006). In the last chapter, I developed and applied a mathematical model that quantifies the spatial processes of sea lice transmission from an isolated salmon farm to migrating wild juvenile salmon. While this was an important first step, I did not completely evaluate the conservation implications of sea lice transmission. In many situations, juvenile salmon must migrate past several salmon farms, presumably increasing the exposure to sea lice. Further, data on sea lice transmission needs to be combined with data on sea lice pathogenicity to evaluate the resulting juvenile salmon mortality. In this chapter, I combine the transmission model with a pathogenicity model to analyze data from both louse surveys of juvenile salmon migrating past multiple farms and observations on the survival of naturally infected juvenile salmon collected from the same populations in the same years and reared in ocean enclosures. The model synthesizes several paired transmission and survival datasets to estimate sea lice transmission as well as juvenile salmon mortality.

4.2 Methods

For 2 years (2004-2005), I studied sea lice abundances on juvenile pink and chum salmon as they migrated past active salmon farms, each containing $\approx$ 600,000 Atlantic salmon (*Salmo salar*). There were three migration routes containing two, two, and three farms, which I studied for 40, 60, and 80 km, and labeled as Kingcome

*The methods and results of this chapter have been published in Krkošek, M., Lewis, M.A., Morton, A., Frazer, L.N., and Volpe. J.P., 2006. Epizootics of wild fish induced by farm fish. *Proceedings of the National Academy of Sciences of the United States of America.* 103, 15506-15510

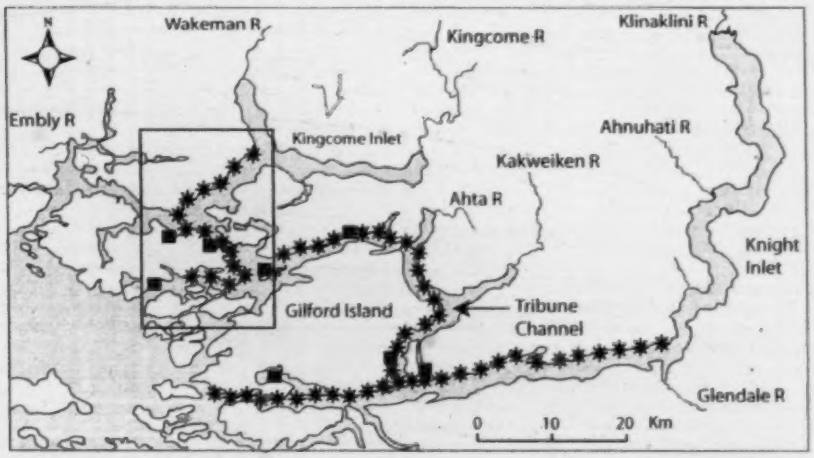

Figure 4.1: Study area and sample sites (stars) for the Kingcome Inlet data sets collected in 2005 (boxed area) and the Tribune Channel and Knight Inlet data sets collected in 2004. Active salmon farms under study are identified by filled squares. Fallow and smolt farms are not shown. Gilford Island is situated east of northern Vancouver Island, BC, Canada.

Inlet (KC), Knight Inlet (KN), and Tribune Channel (TR), respectively (Figure 4.1). As the salmon approached and passed the salmon farms, I sampled them at 1- to 3-km intervals, counting sea lice on $\approx$ 100 juvenile salmon at each site using the non-destructive sampling method described in Chapter 2. At each site I caught the juvenile salmon with a beach seine (45 m long by 6 m deep with 5 mm knotless mesh bunt) and retained a random subsample alive in buckets. In 2004, I examined $\approx$50 juvenile pink and $\approx$50 juvenile chum salmon at each site. In 2005, I examined 50–80 juvenile pink salmon at each site. In 2005, I differentiated chalimus lice between chalimus I/II and chalimus III/IV stages (Figure 1.2). I resolved the species distribution of lice using data from a parallel lethal sampling program conducted by A. Morton who collected weekly samples of 30 juvenile pink and 30 juvenile salmon near the north-west end of Tribune Channel using similar beach seine methods.

The transmission data set totaled 14,255 juvenile salmon nonlethally assayed for copepodid, chalimus, and motile stage lice at 1- to 3-km intervals along 40–80 km of three different migration routes containing two to three farms each (Fig. 4.1). In 2004, I collected three replicate data sets along the 80 km Tribune Channel transect during the dates April 18–28, April 28 to May 8, and May 21–29. These data sets are labeled TR-I, TR-II, and TR-III, respectively. Temperature and salinity averaged 9.0°C and 30.2ppt (TR-I), 10.4°C and 26.1ppt (TR-II), and 12.3°C and 22.2ppt (TR-III). Also in 2004, I collected three replicate data sets of the 60 km Knight

Inlet transect during the dates April 20-25, May 2-10, and May 21-31. These data sets are labeled KN-I, KN-II, and KN-III, respectively. Temperature and salinity averaged 8.8°C and 30.4ppt (KN-I), 9.9°C and 25.8ppt (KN-II), and 12.0°C and 21.1ppt (KN-III). In 2005 I collected two replicate data sets of the 40 km Kingcome Inlet transect during April 17-23 and April 22-25. These data sets are labeled KC-I and KC-II, respectively. Temperature and salinity averaged 7.8°C and 25.8ppt (KC-I) and 11.5°C and 22.5ppt (KC-II). Most of the motile lice were *L. salmonis* rather than *C. clemensi* (615 of 653 were *L. salmonis* in 2005 and 576 of 586 were *L. salmonis* in 2004).

4.3 Transmission model

I model the juvenile salmon migration routes as a one-dimensional infinite domain upon which juvenile salmon migrate and experience sea lice infection dynamics given by the delay differential equations

$$\begin{aligned} \frac{dC}{dx} &= \frac{\beta}{v}[L(x) - L(x-\lambda_c)] \\ \frac{dH}{dx} &= \frac{s_c\beta}{v}[L(x-\lambda_c) - L(x-\lambda_h], \\ \frac{dm}{dx} &= \frac{s_c s_h\beta}{v}[L(x-\lambda_h) - L(x-\lambda_m], \end{aligned} \tag{4.1}$$

which track the mean abundances of copepodid (C), chalimus (H), and motile (M) lice, respectively. This is the same transmission model I described in Chapter 3, only expressed as differential rather than integral equations. The advantage of writing the model this way is that it will be easier to see the link to the salmon survival model I present below. Salmon migrate at an average velocity, v, and encounter local densities of infectious planktonic copepodids (L), which then attach to host fish at rate β. The proportions of surviving copepodids and chalimi are s_c and s_h, respectively. The λs are the cumulative distances salmon travel during successive louse developmental stages (C, H, and M). The model has the same advection-diffusion submodel for larval dispersion as described in section 3.3.1.

To fit the model I first imposed some constraints on the parameters. I fixed $\gamma = 1.56$ km $\cdot$ day^{-1}, the average seaward advective flow for the Broughton Archipelago (Brooks, 2005). I also fixed $\mu_n = 4/5$ days^{-1} and $\mu_c = 1/5$ according to experimental data of larval developmental and survival rates (Johnson and Albright, 1991b). Pink and chum datasets shared four parameters (larval dispersion, louse demographic rates, and ratios of farm and ambient louse production rates), because pink and chum salmon data were collected simultaneously (there is no basis for a difference in these parameter values between host species). These common parameters were the diffusion coefficient of louse dispersion (D), ratios of source

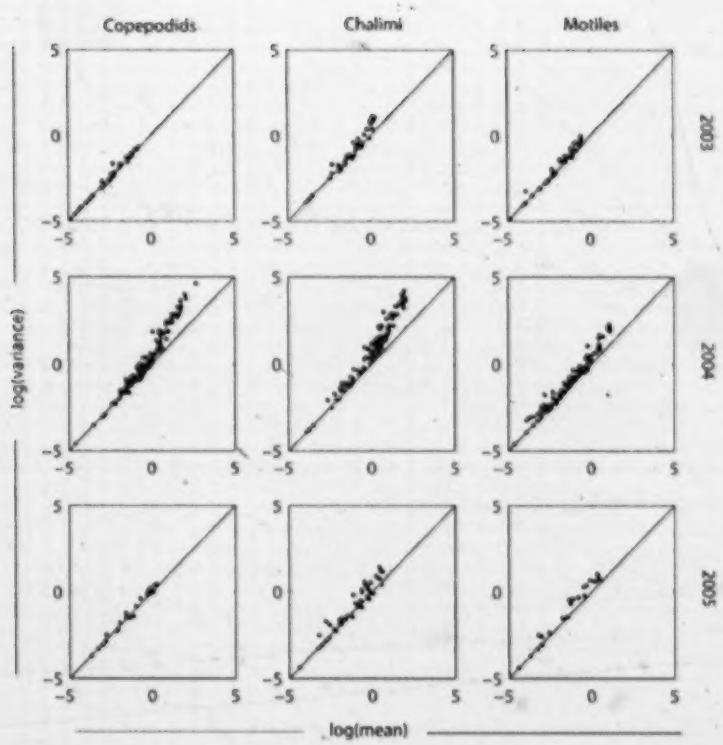

Figure 4.2: Log variance vs. log mean for the three parasitic stages of sea lice infecting juvenile pink and chum salmon in the Broughton Archipelago in British Columbia from 2003-2005. Clustering of samples along the 1:1 variance to mean line is characteristic of a Poisson infection process. There are 41, 117, and 29 samples of $\approx$ 100 juvenile salmon from 2003, 2004, and 2005, respectively. Compare with figure 5 in Shaw and Dobson (1995).

strengths (ϕ_1/κ; the subscript denotes the farm number), and the ratios of the mean durations of louse development stages (λ_h/λ_c and λ_h/λ_m). The host species-specific parameters were allowed to vary between host species. These parameters were louse survival (s_c, s_h), the mean distance salmon travel in the mean duration of the parasitic copepodid stage (λ_c), the ambient infection pressure ($\kappa\beta \cdot \nu^{-1}$), and if gravid females were present in the datasets, the average reinfection intensity that motile lice impose ($\varphi\beta \cdot \nu^{-1}$).

The likelihood function consisted of the product of probabilities of observed copepodid, chalimus, and motile counts on each fish of both species across all sample sites within a dataset. I used the same stochastic formulation I used in Chapter 2, where each development stage was Poisson distributed with a mean given by equations 4.1. The assumption of a Poisson error distribution is supported by the low level of dispersion seen in the data (Figure 4.2). That is, if Θ is the set of parameters common to pinks and chums, and if Δ_i is the set of parameters specific to pinks ($i = p$) or chums ($i = c$), then the likelihood function is

$$\prod_{s} \prod_{i=p} \prod_{j=c,h,m} \prod_{k_{s,i}} P\{N_{i,j} = n_k | \Theta, \Delta_i\}, \tag{4.2}$$

where s indexes the number of sample sites in a dataset, i indexes the host species (pink or chum), j indexes the developmental stage (copepodid, chalimus, motile), and $k_{s,i}$ indexes the number of fish of species i in sample s. The maximum-likelihood values of the six shared and five to six species-specific parameters were estimated using the genetic algorithm toolbox in Matlab. Several optimizations were run on each dataset until the optimum was consistently found.

I fit three different models to the data. The models consisted of only ambient-origin lice, farm-origin lice, and both. The model with only ambient-origin lice was nested within the model with both sources, permitting me to use a likelihood ratio test to test the null hypothesis that lice from farms do not infect wild salmon (all lice are ambient origin). Because not all the models were nested, we used Akiake Information criteria to select the best model from among the three posed.

4.4 Survival data

The survival data used in the 2004 study were collected by Alexandra Morton and published in Morton and Routledge (2005). Morton collected naturally infected juvenile pink and chum salmon from the Broughton Archipelago and reared them in ocean enclosures. The enclosures were 139 L and 189 L plastic barrels with 3 mm plastic mesh covering the two ends. She collected the fish, sorted them into infection categories using the nonlethal technique described in Chapter 2, and distributed the

fish among replicate and randomized barrels within each category. She then fed the fish commercial salmon feed every two hours for several weeks and monitored the survival of the fish. For further details of her study I refer the reader to her publication, Morton and Routledge (2005). Morton conducted three replicate trials, but I used her second trial for pinks and chums, only. I excluded the first trial because the initial sea lice abundances were too low to be informative. I excluded the third trial because of probable confounding effects of increased temperature and reduced salinity (Morton and Routledge, 2005).

In 2005, I conducted a survival study similar to Morton and Routledge (2005), but with some important differences. I did not examine the fish for sea lice before the trial but rather used salmon collected from several locations along a gradient of sea lice abundances. The gradient corresponds to the salmon's passage through the zone of salmon farms I studied in 2005. I used the same flow through enclosures as in the 2004 study (Morton and Routledge, 2005) but situated the experimental site at a different location towards the western end of the study area. I chose the location for stable oceanographic conditions (temperature and salinity remained within 8–12°C and 28–32ppt) and maximal distance from salmon farms and wild salmon migration routes to prevent new infections (only 1 copepodid and 22 chalimus I/II lice were observed on 2,423 surviving salmon). I transported the fish from their source location in aerated buckets and then transferred them into the enclosures with a 15 × 15-cm dip net. I fed the fish commercial salmon feed and also checked for dead and dying fish every 2 hours during daylight. I endeavored to remove and examine moribund fish before death in order to minimize the chance that motile lice may leave a dead host.

4.5 Survival models

The time-series analysis of mortality events consisted of a likelihood based comparison of survival models that described how lice change in pathogenicity as they mature. Lice probably change in pathogenicity as they mature because they become much larger (Figure 1.3 d) and so may feed more intensely on host surface tissues. In particular, the transition from chalimus to motile stages (Figures 1.2-1.3) marks a significant transition in size, movement, and foraging behaviour of the lice (Pike and Wadsworth, 2000).

In the survival analysis, $Q(t)$ is the probability a host fish survives to time t. The probability density function of mortality events is

$$f(t) = \frac{d}{dt}[1 - Q(t)], \tag{4.3}$$

and, because the data were censored (the experiments ended before the fates of all

fish were observed), the likelihood function is

$$\prod_i f(\tau_i) \prod_j Q(\tau_j) \tag{4.4}$$

where the τ_i are the mortality times for each dead fish, and the τ_j are the times each surviving fish was removed from its enclosure and released. The likelihood function includes all treatment levels and their replicates.

4.5.1 Analysis of 2004 survival data

The initial conditions consisted of copepodids and chalimus I/II stage lice, which are much smaller and probably less pathogenic than older and larger developmental stages. I considered two survival models that reflect changes in pathogenicity as lice progress through development and growth. Because the control treatments (no lice) experienced very low mortality (in four treatments with 60 fish each, there were 2, 2, 2, and 1 mortalities), I exclude natural mortality from the models. The first model assumes lice initially have no impact but increase in pathogenicity later in their developmental sequence. The second model assumes lice have an initial impact and then transition into a more pathogenic stage. I will describe the second model, of which the first model is a special case. Simple models where louse pathogenicity was constant through chalimus and motile stages were not considered because of the large change in parasite size and feeding behaviour between chalimus and motile stages.

I approximate the change in pathogenicity by dividing the louse life cycle into two stages. The first pathogenic stage begins with chalimus lice, which induce mortality in their host at rate α_1 per parasite per unit time. The second stage of increased pathogenicity induces host mortality at rate α_2 per parasite per unit time. I leave the waiting time between these stages to be a free parameter, allowing me to identify where in the parasites life cycle there is a marked change in pathogenicity. I also leave the variance in this waiting time to be a free parameter by dividing the first pathogenic stage into a series of n substages, each of equal pathogenicity and with exponentially distributed waiting times of equal duration. The waiting time therefore has a gamma distribution, ψ, with mean μ^{-1} and variance $(n\mu)^{-1}$ (Lloyd, 2001; Keeling and Grenfell, 2002). The probability that a louse remains in the first pathogenic stage after t time units is

$$\xi(t) = 1 - \int_0^t \psi(\tau) d\tau \tag{4.5}$$

Assuming the second stage persists over the time scale of the observational studies, the probability that a fish carrying H_0 young chalimus lice at time 0 is alive at time t is expressed by the survival function

$$Q(t) = \exp\left[-H_0 \int_0^t \Lambda(\tau) d\tau\right] \tag{4.6}$$

where $\Lambda(\tau) = \alpha_1 \xi(\tau) + p\alpha_2 [1 - \xi(\tau)]$ is a variable hazard rate determined by the progression of lice from one pathogenic stage to the next. Here p is the proportion of lice that survive to reach the second stage. There are four parameters (α_1, $p\alpha_2$, n, μ) to be estimated. The first model, where there is no initial mortality, occurs when $\alpha_1 = 0$.

4.5.2 Analysis of 2005 survival data

The survival analysis of 2005 data required a different formulation because initial lice abundances were spread across developmental stages rather than all lice being young chalimus stage as occurred in the previous section. An immediate decline in motile lice was observed after stocking fish in the observational vessels (motile lice are easily dislodged and can freely leave and swim in search of new hosts). The initial abundances of lice were largely chalimus III/IV stage lice and motile lice (some of which were likely dislodged due to fish handling). I assumed, based on the known longevity of motile lice (Johnson and Albright, 1991b), that after the initial loss of motile lice, the abundance of motile lice remained roughly constant for the remainder of the experiment. The data were first sorted by the number of motile lice each fish carried at death or termination of the experiment. In addition, I assumed pathogenicity remained roughly constant from the chalimus stage III/IV lice onward. The probability of host survival to time t is

$$Q(t) = \exp[-\alpha m t], \tag{4.7}$$

where the constant hazard rate αm which is the number of motile lice each fish carried times the rate of mortality each motile louse imposes on its host (α). The likelihood function capturing all fish mortality and live release events is then given by equation 4.4

4.6 Connecting transmission and survival models

To estimate the cumulative mortality of outmigrating juvenile salmon caused by sea lice, I coupled the survival model to the transmission model. The models are linked by the chain rule, which maps time onto space by the mean migration velocity of juvenile salmon, $v = x \cdot t^{-1}$. That is, any function describing the dynamics of salmon (or parasitic lice) in time $g(t)$ becomes a function of space $g(x)$ by applying the chain rule $dg/dx = dg/dt \cdot dt/dx = v^{-1} \cdot dg/dt$.

For the 2004 data and models, the spatial model for the dynamics of lice

developing through the pathogenic stages is

$$\begin{aligned}
\frac{dP_{1,1}}{dx} &= \frac{p_c\beta}{v}L(x-\lambda_h) - \frac{1}{v}(n\mu_1+\alpha_1)P_{1,1} \\
\frac{dP_{1,2}}{dx} &= \frac{n\mu_1}{v}P_{1,1} - \frac{1}{v}(n\mu_1+\alpha_1)P_{1,2} \\
&\vdots \\
\frac{dP_{1,n}}{dx} &= \frac{n\mu_1}{v}P_{1,n-1} - \frac{1}{v}(n\mu_1+\alpha_1)P_{1,n} \\
\frac{dP_2}{dx} &= \frac{n\mu_1}{v}P_{1,n} - \frac{\sigma}{v}P_2
\end{aligned} \tag{4.8}$$

The first term in the first equation describes the influx of chalimus stage lice, similar to the transmission model. These lice then move through successive pathogenic substages, the number of which was estimated in the survival analysis. $1/\mu_1$ is the mean duration of the first pathogenic stage, which has variance $(n\mu)^{-1}$. Once arriving in the second pathogenic stage, lice die at rate σ, which represents the sum of natural parasite mortality and parasite-induced host mortality rates ($\sigma = \mu_2+\alpha_2$), which were not separately identifiable. However, σ could be estimated directly from the transmission dynamics data as $\sigma = v \cdot (\lambda_m - \lambda_h)^{-1}$. The proportion of juvenile salmon at location x surviving sea lice infestation is then determined by

$$\frac{dN}{dx} = -\frac{1}{v}\left[\alpha_1 \sum_{i=1}^{n} P_{1,i}(x) + p\alpha_2 P_2(x)\right] N, \tag{4.9}$$

where $N(x_0) = 1$ and x_0 is the landward extreme of the study area. There are four parameters (α_1, $p\alpha_2$, n, μ_1) that were estimated from the survival data and two parameters ($\beta p_c \cdot v^{-1}, \sigma$) estimated from the transmission data.

The cumulative mortality of outmigrating juvenile salmon in 2005 was calculated in a similar fashion using the chain rule. In this case, the survival model was much simpler (no substages with low pathogenicity) and was simply the solution of

$$\frac{dN}{dx} = -\frac{\alpha m(x)}{v}N \tag{4.10}$$

where $m(x)$ is the spatial distribution of motile lice as estimated by the transmission dynamics model.

4.7 Results

Across all the transmission datasets, the statistics show that farm salmon infected wild salmon with sea lice (Table 4.3), that the best model contained both farm and ambient sources of lice (Table 4.2), and that the farms each produced several orders of magnitude more lice than ambient levels (Table 4.4) . Gravid female lice occurred in the TR-III and KN-III data sets and so the models of these data sets contain louse reproduction. The model that contains both ambient and farm sources of lice fit the data well (Figures 4.3, 4.4, 4.5, 4.6 and 4.7). With the parameter estimates from

Salmon	$\alpha_1, day \cdot lice^{-1}$	$p\alpha_2, day \cdot lice^{-1}$	n	μ_1^{-1}, days
Pink	0.0078	0.0011	171	17
Chum	0.0011	0.0041	11	36

Table 4.1: Parameter estimates of the best-fit survival model to 2004 pink and chum data. The mean time to the onset of increased pathogenicity is μ_1^{-1}, and the variance in this onset is $(n\mu_1)^{-1}$

the best-fit model (Table 4.4), I reconstructed the spatial distributions of infective larvae originating from each source. Farm salmon were the primary source of lice, raising the density of infective parasite larvae above ambient levels for > 80 km of the migration route (Figures 4.3 -4.7).

The data for the 2004 survival experiments totaled 3,687 juvenile salmon with initial infection intensities ranging from zero to five chalimus lice. The data best supported a survival model that contained a gamma-distributed random variable for the parasites developmental stage at which there is a marked increase in pathogenicity (likelihood ratio test; pinks, $P = 5 \times 10^{-18}$ and chums, $P = 5 \times 10^{-35}$; Figures 4.8 and 4.9 and Table 4.1). The simpler model of the 2005 survival data, fit the data well, but it underestimated the mortality of heavily infected hosts (Fig. 4.10). The estimated value of α was 0.02 (day · lice)$^{-1}$. If we equate α with α_1 estimated in the survival analysis of 2004 pink salmon we see that $p = 0.05$, which is lower than the estimates of chalimus lice survival (s_h) in the analysis of lice transmission dynamics (Table 4.4).

I mapped the survival models onto space using the chain rule and the average juvenile salmon migration speed ($\approx$ 1 km · day^{-1}; Table 4.4) and then coupled it to the larval distributions and infection rates identified by the transmission dynamics model. By removing ambient lice from the best-fit model, I calculated the proportions of the juvenile salmon populations that survived parasitism from farm origin lice. These were 5–26% for pink salmon and 10-70% for chum salmon in the Tribune Channel data sets, 49–78% for pink salmon and 69–91% for chum salmon in the Knight Inlet data sets, and 11–35% for pink salmon in the Kingcome Inlet data sets. Both data sets and models for 2004 and 2005 studies yielded similar estimates of sea lice transmission and wild juvenile salmon mortality.

4.8 Discussion

In this chapter I have used data and models to link sea lice transmission and wild juvenile salmon survival. The data sets were extensive, totaling nearly 20,000 juvenile salmon. The transmission data consisted of sea lice abundances on juvenile pink and chum salmon migrating past salmon farms. The survival data consisted of

	Ambient		Farm		Ambient plus Farm	
Dataset	$\triangle AIC$	w_j	$\triangle AIC$	w_j	$\triangle AIC$	w_j
TR-I	3606	$\to 0$	48	4.6×10^{-11}	0	$\to 1$
TR-II	3336	$\to 0$	120	8.8×10^{-27}	0	$\to 1$
TR-III	6380	$\to 0$	214	3.4×10^{-47}	0	$\to 1$
KN-I	2088	$\to 0$	32	1.1×10^{-7}	0	$\to 1$
KN-II	1524	$\to 0$	121	6.5×10^{-27}	0	$\to 1$
KN-III	1652	$\to 0$	38	6.8×10^{-9}	0	$\to 1$
KC-I	1444	$\to 0$	26	2.3×10^{-6}	0	$\to 1$
KC-II	1093	$\to 0$	200	4.1×10^{-44}	0	$\to 1$

Table 4.2: Model selection statistics ($\triangle$ AIC and Akaike weights, w_j) for each model fit to each dataset. Models are defined by their lice-source components: ambient only, farm only, and ambient plus farm. ($w_j \to 0$ means $w_j < 1 \times 10^{-234}$, and $w_j \to 1$ means $w_j > 0.9999999$).

Year	Dataset	R	DF	P
2004	TR-I	3,620	7	$\to 0$
	TR-II	3,350	7	$\to 0$
	TR-III	6,396	8	$\to 0$
	KN-I	2,068	6	$\to 0$
	KN-II	1,536	6	$\to 0$
	KN-III	1,666	7	$\to 0$
2005	KC-I	1,458	7	$\to 0$
	KC-II	1,107	7	$\to 0$

Table 4.3: Likelihood ratio tests of the null hypothesis that farm salmon do not infect wild salmon. R is the likelihood ratio statistic, DF is the degree of freedom (difference in the number of parameters between nested models: ambient lice sources only and ambient plus farm sources), and P is the associated P value ($P \to 0$ means $P < 1 \times 10^{-234}$).

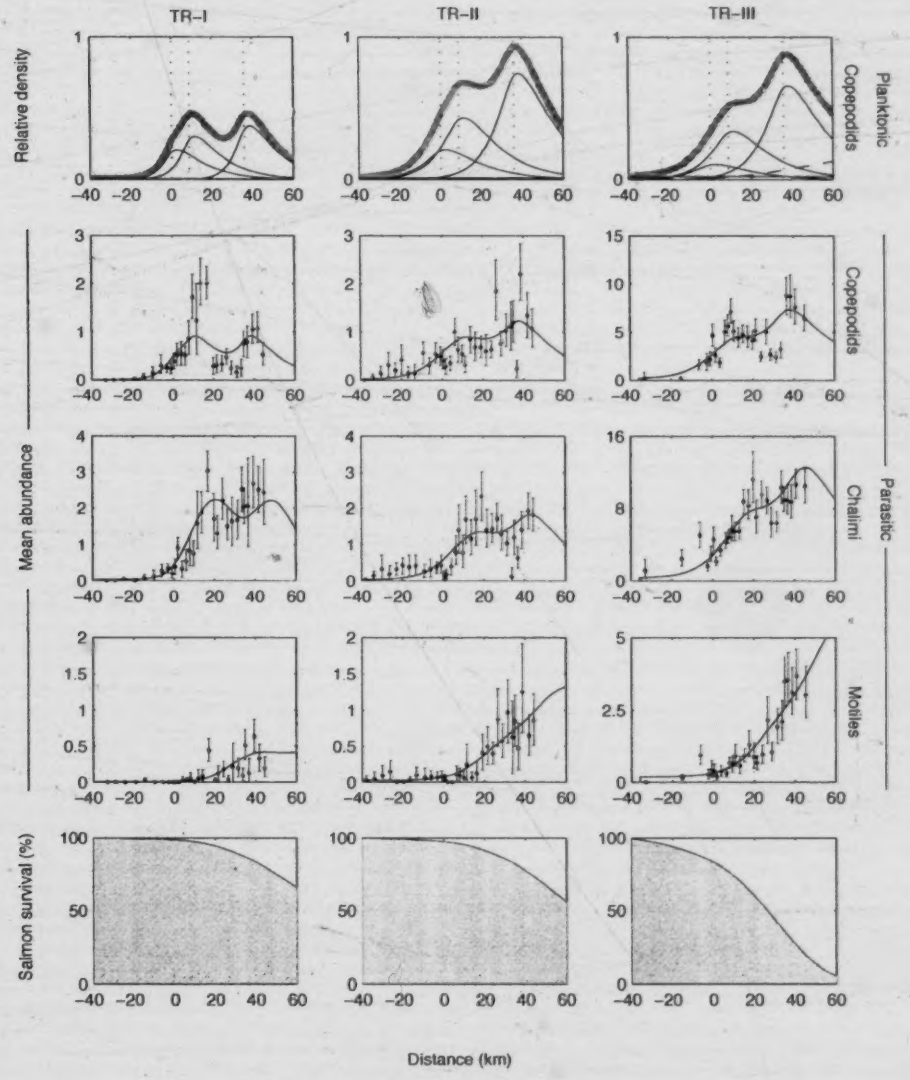

Figure 4.3: Sea lice transmission and survival of juvenile chum salmon migrating past three active salmon farms in Tribune Channel. The seaward migration of salmon is from left to right, and the farm locations are shown by vertical dotted lines in the first row. The three replicate data sets were collected along the Tribune Channel migration corridor in 2004 (see Figure 4.1). The first row shows the estimated spatial distributions of planktonic copepodids originating from all sources (thick gray line), from farm salmon (three thin curves), from ambient sources (horizontal thin line), and the second generation of farm-origin lice (dashed curve, TR-III only). The middle three rows depict the mean abundances of lice (±95%bootstrap confidence interval) and maximum-likelihood model fits (black lines) along the migration route for the developmental progression through parasitic copepodid, chalimus, and motile stages. The bottom row depicts the estimated remaining juvenile salmon that survived sea lice infestation.

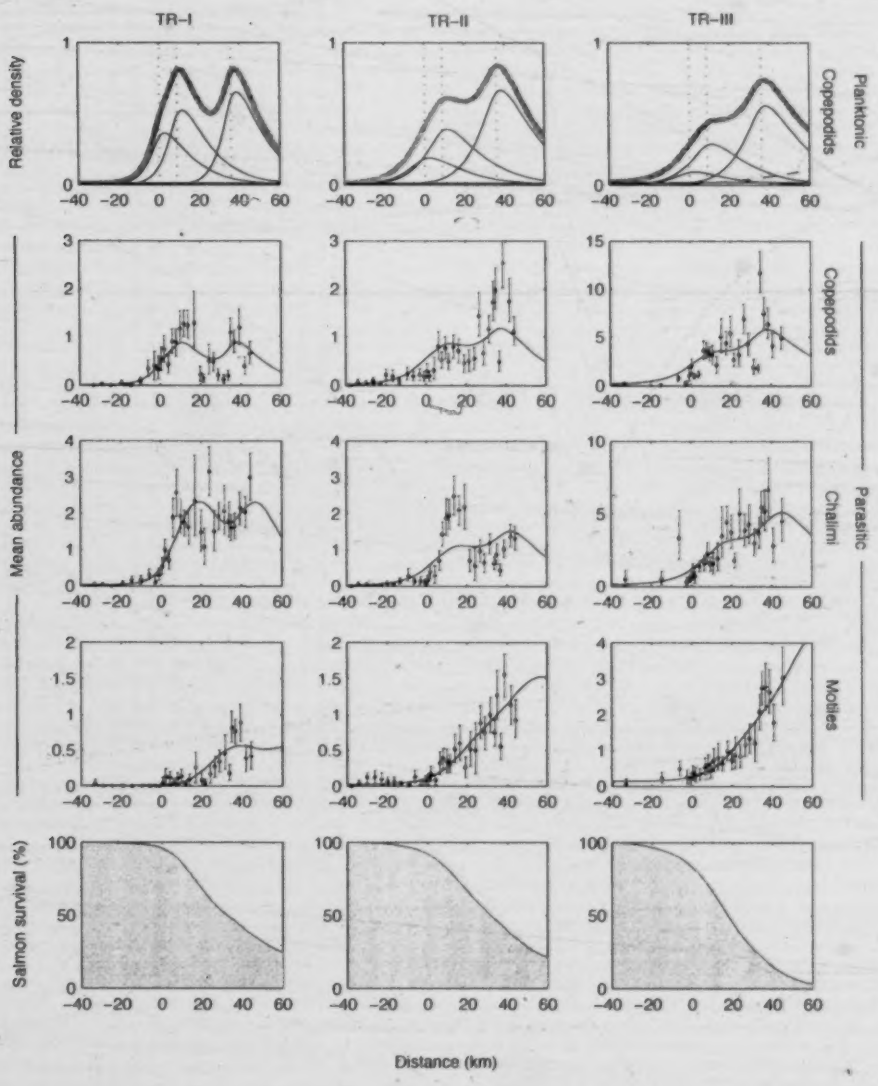

Figure 4.4: Sea lice transmission and survival of juvenile pink salmon migrating past three active salmon farms in Tribune Channel. The seaward migration of salmon is from left to right, and the farm locations are shown by vertical dotted lines in the first row. The three replicate data sets were collected along the Tribune Channel migration corridor in 2004 (see Figure 4.1). The first row shows the estimated spatial distributions of planktonic copepodids originating from all sources (thick gray line), from farm salmon (three thin curves), from ambient sources (horizontal thin line), and the second generation of farm-origin lice (dashed curve, TR-III only). The middle three rows depict the mean abundances of lice (±95%bootstrap confidence interval) and maximum-likelihood model fits (black lines) along the migration route for the developmental progression through parasitic copepodid, chalimus, and motile stages. The bottom row depicts the estimated remaining juvenile salmon that survived sea lice infestation.

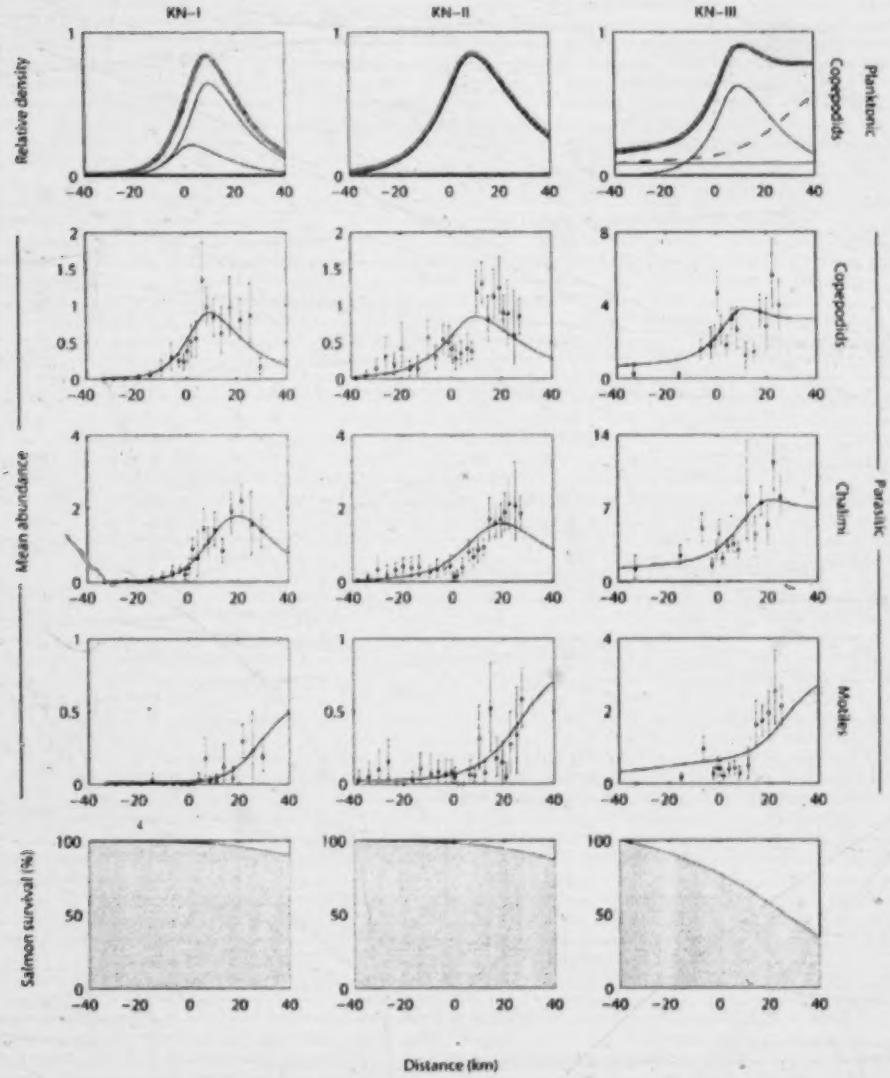

Figure 4.5: Sea lice transmission dynamics and mortality impact on juvenile chum salmon migrating past two active salmon farms (vertical dotted lines in the first row) in the Knight Inlet migration corridor (Fig. 4.1). The seaward migration of salmon is from left to right. The first row shows the estimated spatial distributions of planktonic copepodids originating from all sources (thick gray line), from farm salmon (two thin curves), and from ambient sources (horizontal thin line) and the second generation of farm origin lice (dashed curve, KN-III only). Reproduction of lice parasitizing the juvenile salmon was not considered in KN-I and -II due to the absence of gravid female lice in those datasets. The middle three rows depict the mean abundances of lice (95% bootstrap confidence interval) and maximum-likelihood model fits (black lines) along the migration route for parasitic copepodids, chalimi, and motiles. The bottom row depicts the estimated remaining juvenile salmon population that survived sea lice infestation.

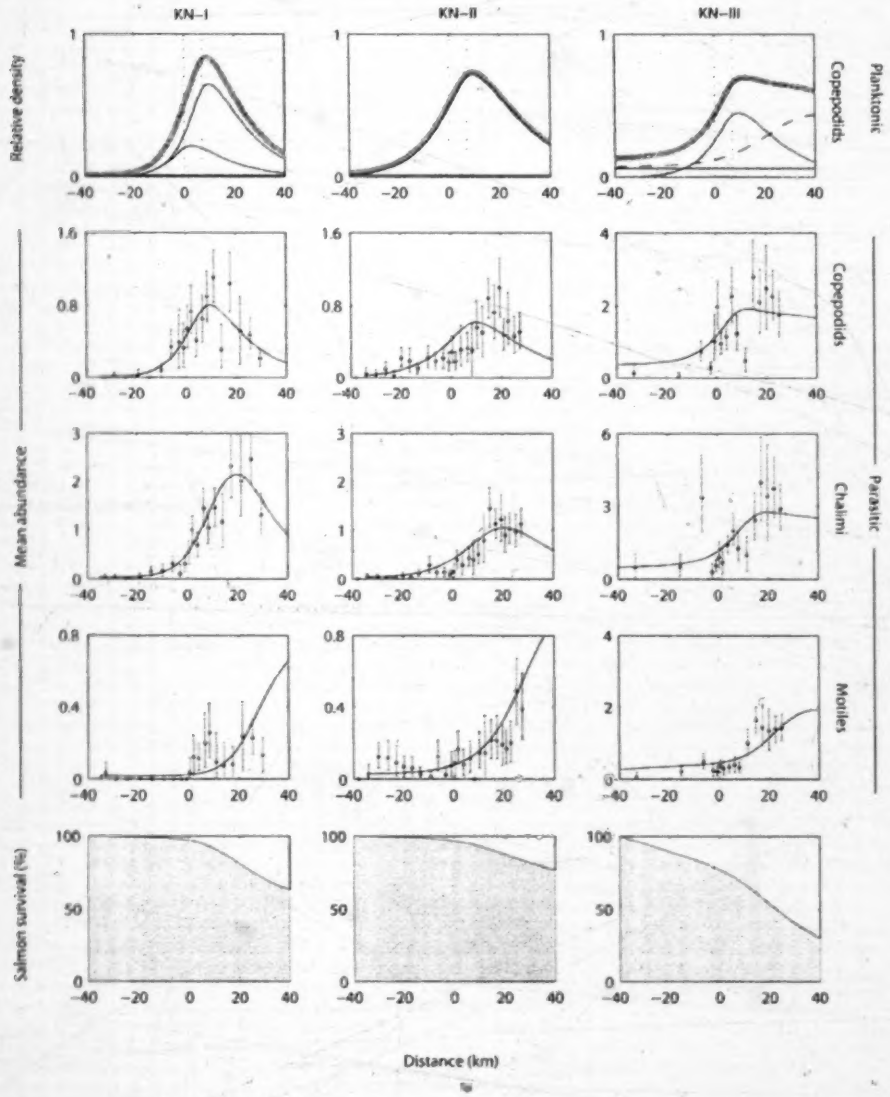

Figure 4.6: Sea lice transmission dynamics and mortality impact on juvenile chum salmon migrating past two active salmon farms (vertical dotted lines in the first row) in the Knight Inlet migration corridor (Fig. 4.1). The seaward migration of salmon is from left to right. The first row shows the estimated spatial distributions of planktonic copepodids originating from all sources (thick gray line), from farm salmon (two thin curves), and from ambient sources (horizontal thin line) and the second generation of farm origin lice (dashed curve, KN-III only). Reproduction of lice parasitizing the juvenile salmon was not considered in KN-I and -II due to the absence of gravid female lice in those datasets. The middle three rows depict the mean abundances of lice (95% bootstrap confidence interval) and maximum-likelihood model fits (black lines) along the migration route for parasitic copepodids, chalimi, and motiles. The bottom row depicts the estimated remaining juvenile salmon population that survived sea lice infestation.

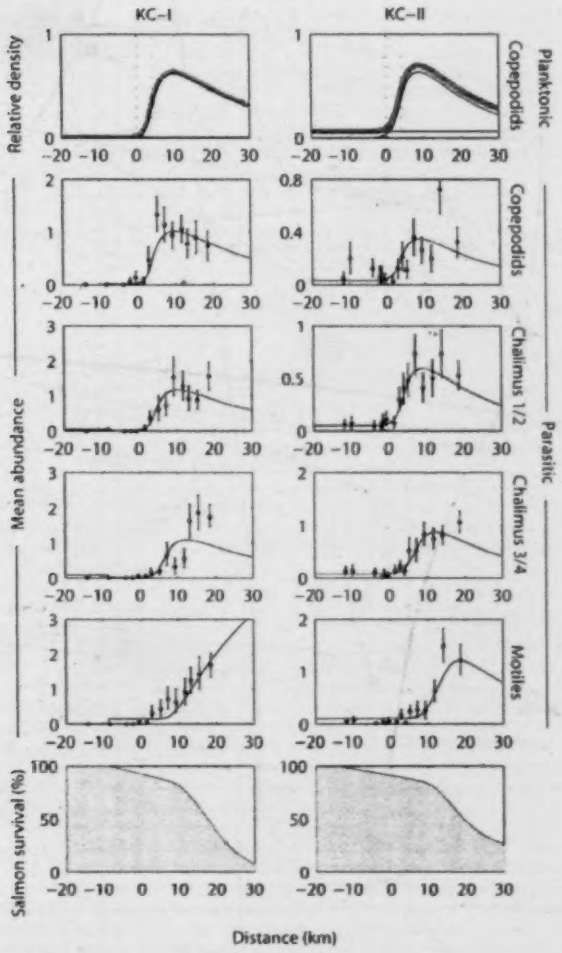

Figure 4.7: Sea lice transmission dynamics and mortality impact on juvenile pink salmon migrating past two active salmon farms (vertical dotted lines in the first row) in the Kingcome Inlet migration corridor (Fig. 4.1). The seaward migration of salmon is from left to right. The first row shows the estimated spatial distributions of planktonic copepodids originating from all sources (thick gray line), from farm salmon (two thin curves), and from ambient sources (horizontal thin line). Reproduction of lice parasitizing the juvenile salmon was not considered due to the absence of gravid female lice. The middle three rows depict the mean abundances of lice (95% bootstrap confidence interval) and maximum-likelihood model fits (black lines) along the migration route for parasitic copepodids, chalimi, and motiles. The bottom row depicts the estimated remaining juvenile salmon population that survived sea lice infestation.

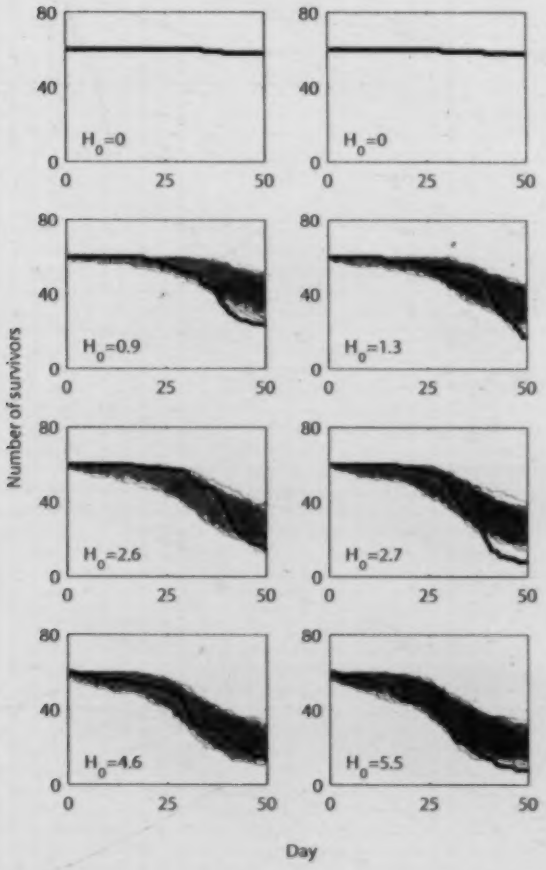

Figure 4.8: Survival of juvenile chum salmon over a range of sea lice abundances. Sixty juvenile chum salmon initially infested with H_0 lice (all copepodids or chalimus I/II) were introduced into flow-through ocean enclosures and provisioned with salmon feed. Each image corresponds to an individual enclosure. The black line shows the trajectory for the daily number of survivors. The light-gray lines are the trajectories of 1,000 simulations of the best-fit model. The model was simulated as a Markov chain tracking the number of survivors in time. Each day, the number of mortalities was drawn from the number of survivors on the previous day using a binomial distribution with mortality probability calculated from the best-fit survival model. For all treatment replicates, the model has the same parameter values, except for H_0, which is specific to each enclosure.

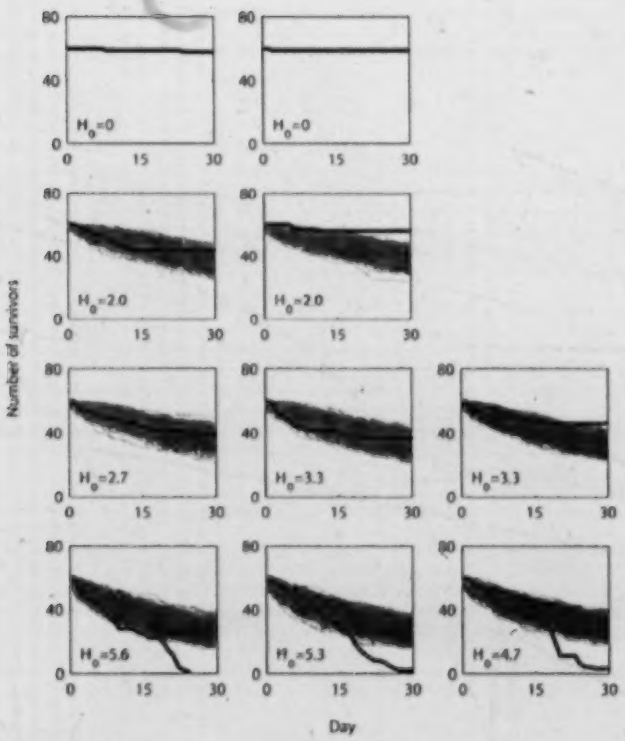

Figure 4.9: Survival of juvenile pink salmon infested with sea lice in 2004. Sixty juvenile chum salmon infested with H_0 lice (all copepodids or chalimus I/II) were introduced into flowthrough ocean enclosures and provisioned with salmon feed. Each image corresponds to an individual enclosure. The black line shows the trajectory for the daily number of survivors. The light-gray lines are the trajectories of 1,000 simulations of the best-fit model. The model was simulated as a Markov chain tracking the number of survivors in time. Each day, the number of mortalities was drawn from the number of survivors on the previous day by using a binomial distribution with mortality probability calculated from the best-fit survival model. The model has the same parameter values for all treatment replicates, except for H_0, which is specific to each enclosure.

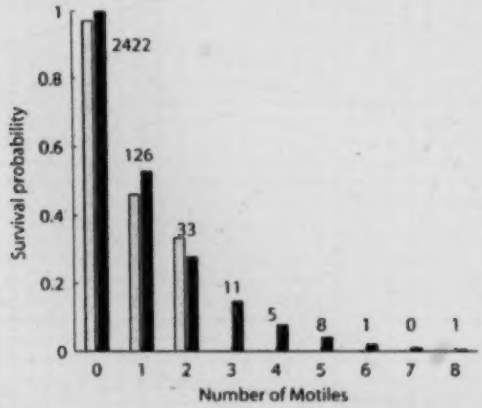

Figure 4.10: Survival of juvenile pink salmon infested with sea lice in 2005. The plot shows the number of survivors in each infection category (light-gray bars) and the best-fit model for the survival probability of these fish after 28 days (the average duration of the observational trials; dark bars). The numbers above each light bar are the number of fish observed in each category.

time series observations on sea lice abundance and fish survival in ocean enclosures. I endeavored to keep the transmission and survival data tightly linked by using for the survival component naturally infected fish collected from the same migration routes and season studied in the transmission component. In this way, the transmission and survival data sets were paired so that the study subjects had the same history of sea lice exposure, food availability, predation pressure, abiotic stressors, etc, prior to capture and data collection. The tight coupling of transmission and survival datasets therefore increases the robustness of the transmission and survival estimates emerging from the analysis of each dataset. The datasets also produce a starting point for generalizing the parameter estimates of the model fitting, but such estimates may vary annually and even within-season, based on changes in the environment.

Although most of the lice observed on the wild juvenile salmon were farm-origin, there were also ambient-origin lice. This was measured in areas landward of the farms where lice abundances were low and spatially uniform. These data can be thought of as a control and conform well to the null models where lice abundances are spatially uniform in the absence of farms. Low abundances of lice have also been observed on juvenile pink and chum salmon during their first months at sea in areas distant from salmon farms (Morton et al., 2004). These lice represent the combined contributions from resident alternate hosts, Chinook salmon (*Oncorhynchus tshawytscha*), sea-run cutthroat trout (*Oncorhynchus clarki*), and

Dolly Varden (*Salvelinus malma*). These species are orders of magnitude less abundant than both farm salmon and returning adult pink and chum salmon, which are the primary natural hosts for lice (Nagasawa, 2001) and are located offshore during the study period (Groot and Margolis, 1991). The different abundances of these hosts mean that farm salmon provide lice a significant novel transmission route, which in this case operates for at least the first 2.5 months of the salmon's marine life (80 km of migration route).

Usually considered benign on adult salmon, *L. salmonis* was a severe pathogen of juvenile pink and chum salmon. Generally, an abundance of more than two motile lice was lethal, and survival of hosts with one or two motile lice was poor (survival of uninfected hosts was nearly 100%. As the lice progressed through their life cycle, they also increased in pathogenicity, but the patterns differed between host species. For pink salmon, the onset of increased pathogenicity occurred abruptly with the emergence of preadult lice, but for chum salmon, it was more widely distributed around adult lice (Table 4.1). The high pathogenicity and abundance of lice resulted in a farm-induced cumulative epizootic mortality of wild juvenile salmon that ranged from 9% to 95%. These results were consistent across multiple data sets spanning temporal, spatial, and taxonomic replication. The estimated mortality of wild salmon is high but consistent with direct field observations of the epizootics, where schools of infested moribund juvenile salmon (Figure 1.3 d) were abundant.

I did not consider the possibility that food limitation or predation risk would be more severe for infected hosts (but see chapter 5). Generally, poor nutrition is thought to reduce the resistance of fish hosts to disease (Blazer, 1992; MacKinnon, 1998), and parasitized prey are known to be more vulnerable to predation (Hudson et al., 1992; Mesa et al., 1998). These interactions would likely increase mortality estimates. Only one assumption, relatively low motile louse mortality, after the initial loss of motile lice due to fish handling, could cause an overestimate of the per-capita impact of lice. However, empirical data suggest motile lice are long-lived (Pike and Wadsworth, 2000), at least as long as their occurrence in the survival trials (16–36 days). It is unlikely that an alternate problem may have predisposed salmon to the epizootics; research programs by universities, conservation organizations, provincial and federal governments, and industry have not identified a prevalent viral or bacterial pathogen or other physical stressor.

The estimated mortality sea lice caused in the wild juvenile salmon populations ranged from moderate to severe. Clearly, an annual and novel parasite induced 95% mortality in wild juvenile salmon cohorts has implications for the conservation of the wild salmon populations. However, there is 85% mortality from marine entry

to spawning for pink salmon in pristine conditions and presumably where juvenile salmon are not exposed to high lice abundances (Heard, 1991; Parker, 1968). The high natural mortality raises the possibility that sea lice, even when abundances are moderately high, may not threaten the wild salmon populations. For example, is there a real threat if 50% of the juvenile salmon are lethally infected but 85% are going to die anyway? To evaluate this one needs to better understand some natural mechanisms in sea lice and Pacific salmon ecology - namely migratory allopatry and compensatory predation. I turn to these issues in the next chapter where I examine sea lice dynamics in an area without salmon farms and then build a mathematical model of sea lice and salmon population dynamics.

	Chums				Pinks				Both Species			
	λ_h, km	$v, km \cdot d^{-1}$	s_c	s_h	λ_h, km	$v, km \cdot d^{-1}$	s_c	s_h	ϕ_1/κ	ϕ_2/κ	ϕ_3/κ	$D, km^2 \cdot d^{-1}$
TR-I	17.3	1.2	0.24	0.13	15.1	1.1	0.25	0.16	8,554	12,407	15,495	11
TR-II	10.4	0.7	0.18	0.17	8.6	0.6	0.17	0.23	5,987	12,425	21,308	25
TR-III	13.5	1.0	0.15	0.15	12.7	0.9	0.10	0.27	1,968	6,176.3	11,989	27
KN-I	18.7	1.3	0.15	0.19	18.0	1.3	0.21	0.20	5,877	17,754	-	16
KN-II	17.3	1.2	0.13	0.25	18.5	1.3	0.13	0.50	60	21,445	-	36
KN-III	16.1	1.1	0.20	0.22	12.2	0.9	0.14	0.41	28	2,611	-	21
KC-I	-	-	-	-	4.7	0.3	0.30	0.11	3,400	29,527	-	1.6
KC-II	-	-	-	-	6.4	0.5	0.19	0.26	240	2,595	-	2.2

Table 4.4: Parameter values estimated from the transmission dynamics model for each dataset in 2004. Parameters: λ_h, average distance salmon migrate during parasitic copepodid and chalimus stages; v, the average seaward migration velocity of juvenile salmon (calculated by dividing λ_h by 14 days, approximately the mean duration of copepodid plus chalimus lice) (**?**); ϕ_i, abundance of copepodids released from farm i; κ, ambient density of copepodids; D, diffusion coefficient of planktonic larvae; s_c, survivorship of parasitic copepodids; s_h, survivorship of chalimi.

Chapter 5

Natural mechanisms in sea lice and Pacific salmon ecology*

5.1 Introduction

In this chapter I step back from the focus on sea lice and salmon farms in the Broughton Archipelago and examine two natural mechanisms influencing sea lice and Pacific salmon ecology: migratory allopatry and compensatory predation. Migratory allopatry is the property of migratory fish and invertebrates that juveniles are protected from parasites because juveniles are spatially separated from infected adult hosts. Compensatory predation is the selective predation on infected hosts and is relevant to juvenile salmon because they naturally experience high predation rates. First I present empirical evidence for migratory allopatry in sea lice and pink salmon by analyzing data on sea lice (*C. clemensi* and *L. salmonis*) infecting juvenile pink salmon during their early marine life in an area without salmon farms. Then I develop and analyze a model of pink salmon population dynamics that incorporates louse-induced mortality and compensatory predation of juvenile pink salmon. I use the model to evaluate the importance of migratory allopatry by evaluating the sensitivity of wild pink salmon populations to increased lice exposure of juvenile fish.

5.2 Study system: Chatham Sound

Skeena River pink salmon are among Canada's largest salmon stocks with escapements of 0.2 to 4.8 million spawners and returns of up to 25 million. In early spring (April) juvenile pink salmon emerge from the Skeena River and coastal

*The methods and results of this chapter have been published in Krkošek, M., Gottesfeld, A., Proctor, B. Rolston, D. Carr-Harris, C., Lewis, M.A., 2007, Effects of host migration, diversity, and aquaculture on sea lice threats to Pacific salmon populations. *Proceedings of the Royal Society of London Series B*, 274, 3141-3149.

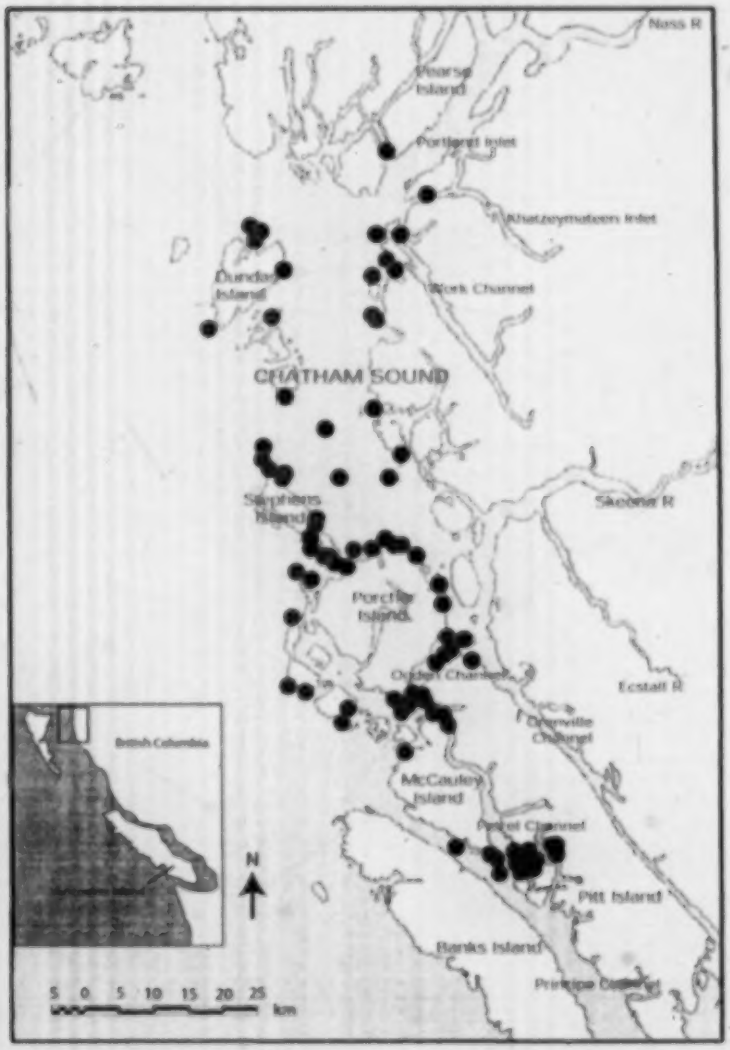

Figure 5.1: Study area and sample locations for 2005. The spatial distribution of sample sites were similar in 2004 and 2006. Chatham Sound and the Skeena River are located on the north coast of British Columbia, Canada. There are no salmon farms in this area.

streams into Chatham Sound (Figure 5.1) where they rear until late summer. There are no salmon farms in Chatham Sound; however the area is experiencing pressure to become a major hub in salmon aquaculture production (Allen Gottesfeld, Skeena Fisheries Commission, pers. com.). During spring there are few adult salmon hosts in these waters and so juvenile and adult salmon are allopatric until late May or June, when the first adult salmon, in this case chinook (*O. tshawytscha*) begin to arrive on their return migration. Abundant returning adult chinook and early runs of coho (*O. kisutch*) appear in July. In contrast, *C. clemensi* has a wide host range (Table 1.1) including locally ubiquitous populations of herring (*Clupea harengus pallasi*), abundant Pacific sandfish (*Trichodon tricodon*) and scattered stickleback (*Gasterosteus aculeatus*) that are sympatric with the juvenile salmon.

5.3 Field Data and Analysis

The sea lice data were generously contributed by Dr. Allen Gottesfeld, Head Scientist of the Skeena Fisheries Commission. Gottesfeld and colleagues studied sea lice infections of juvenile pink salmon in Chatham Sound, British Columbia (Figure 5.1) during the juvenile salmon ocean migration for three years 2004-2006. They sampled from mid to late April through early August in 2004 and 2005 and from mid May to Mid July in 2006. In the early season, they collected juvenile salmon using a dipnet (45 cm diameter with 5 mm knotless mesh on a 2.45 m pole) from a small skiff (4.17m flat-bottomed aluminum skiff). In mid May when the fish reached approximately 55 mm forklength they moved from very shallow (intertidal) habitats to deeper habitats several to tens of meters off the beach. Subsequent to this transition Gottesfeld and colleagues collected juvenile salmon using an Ocean Fish Lift trawl (Holst and McDonald, 2000) towed behind a fiberglass ex-commercial gill net vessel 11 meters in length moving at 2.7-2.9 knots. The trawl net was 5.0 meters wide by 4.6 meters deep and 18.0 meters long. The rigid cod-end of the trawl net minimized damage to live samples, in particular the loss of scales and ectoparasites (Holst and McDonald, 2000). Collections occurred during 1-2 week cruises during which many (though not all) sample sites were fished. Salinity and sea surface temperature were recorded after most collections using a YSI-30 SCT meter. Fish were immediately frozen and labeled for subsequent laboratory analysis. In the lab, individual fish were thawed and assayed for sea lice using a dissecting microscope. Motile stages of sea lice were directly determined to species by their morphological differences (Kabata, 1972; Johnson and Albright, 1991a). Copepodid and chalimus lice were removed from the fish, mounted on permanent slides, and examined under a compound microscope to make species determinations based on detailed morphology (Kabata, 1972; Johnson and Albright, 1991a). I analyzed the data at broad spatial

and temporal scales (i.e. per month over the entire study area). This was necessary because sea lice abundance was too low to support detailed spatial analysis and also to accommodate variation in the spatial distribution of sampling effort among cruises. Differences in sea lice abundance over time and between species were tested using generalized linear models with Poisson error.

5.4 Empirical Results

The three years of surveys resulted in a total of 21,448 juvenile pink salmon assayed for sea lice infections. Of these fish, 13,139 were collected by dipnet and 8,309 were collected by the OFL trawl. There was no effect of gear on sea lice abundance when Gottesfeld and colleagues simultaneously fished both gear types at the same sites. Over the season, the juvenile salmon increased in fork length from 30 mm in early spring up to 130 mm in summer. This corresponds to an increase in weight by two orders of magnitude from about 0.2 g to 20 g. By July most juvenile salmon had well-developed scales but lacked these scales upon marine emergence and for their first 1-2 months of marine life. Throughout the study Gottesfeld and colleagues periodically collected herring (n=143), Pacific sandfish (n=48), and stickleback (n=47) as incidental by-catch in trawls. All these fish species carried *C. clemensi*. The 47 stickleback carried 770 lice total and of the 345 we examined, 223 were *L. salmonis* and 132 were *C. clemensi*. There was one gravid *C. clemensi* observed on the herring, no gravid lice observed on the stickleback, and no motile lice observed on the sandfish. Sea surface temperature ranged from ~10°C in April to ~15°C in August. Sea surface salinity ranged from ~10 ppt near the mouth of the Skeena River to ~32 ppt around the western fringe of Chatham Sound.

The prevalence of *L. salmonis* was generally around 2-3% during the first three months of marine life of pink salmon (Fig. 5.2 and Table 5.1). Come July, there was a marked increase in *L. salmonis* abundance ($p<0.05$). *L. salmonis* prevalence rose to up to 50% in 2004 (Fig. 5.2), and this was due to a general rise in the mean abundance of most louse developmental stages (Table 5.1). Interestingly, this includes an abrupt increase in *L. salmonis* motiles that does not correspond to a preceding developmental progression (Table 5.1). Late June and early July mark the return migration of the first abundant population of adult salmon to these waters, in this case Chinook salmon. These adult salmon are known to carry motile and gravid *L. salmonis* and occur within 10-100 m of abundant populations of juvenile pink salmon (Allen Gottesfeld, pers obs). During this time the juvenile salmon population was predominately distributed through the outer fringe of the study area. In these waters salinity was around 28 ppt, which is suitable for sea lice survival and transmission. The abundance of *L. salmonis* was not significantly related to

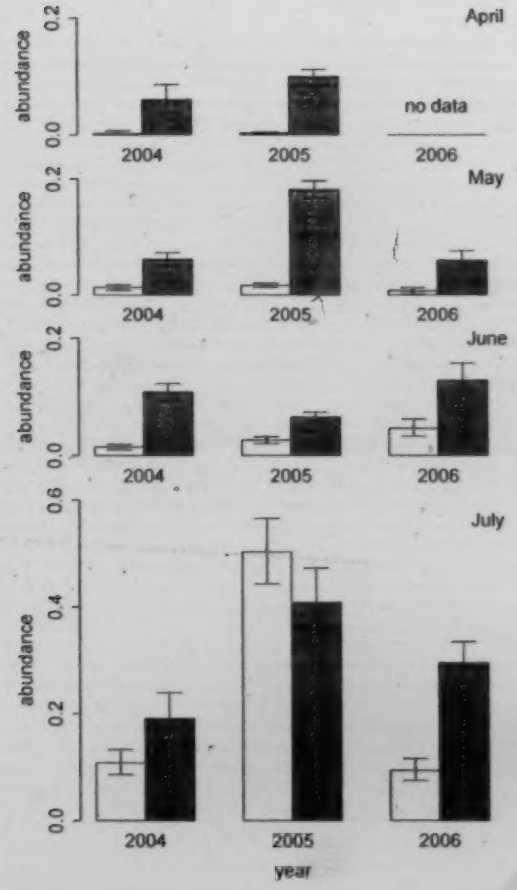

Figure 5.2: Total sea lice abundance (± 95% bootstrap confidence intervals) in spring and summer for *L. salmonis* (light grey) and *C. clemensi* (dark grey) over three years (2004-2006).

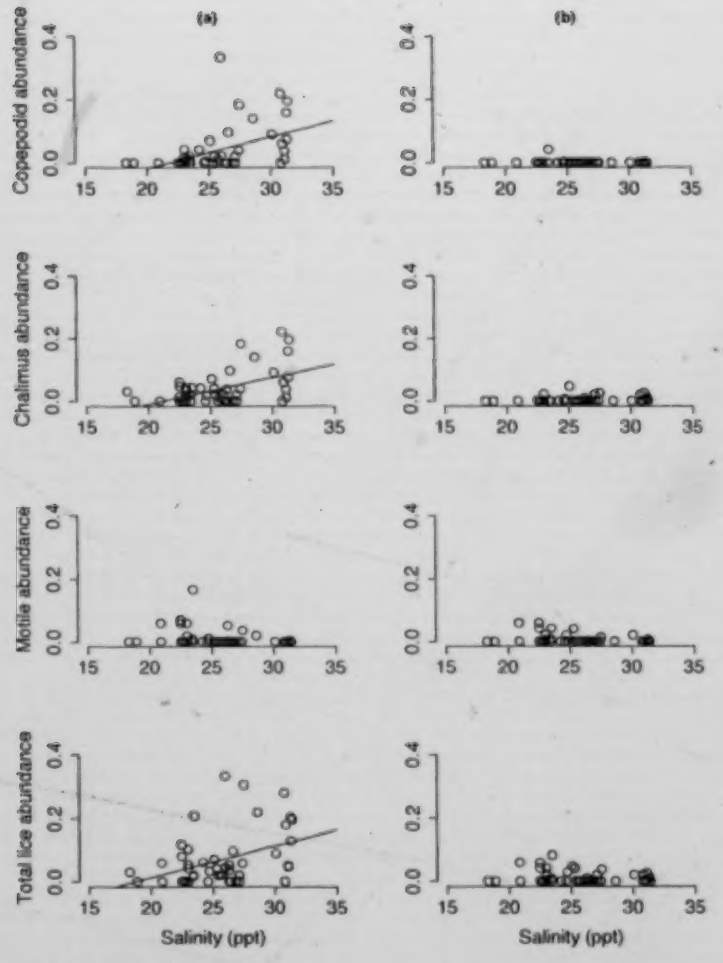

Figure 5.3: Sea lice abundances for (a) *C. clemensi* and (b) *L. salmonis* plotted against salinity for collections of pink salmon in 2004 when salinity was measured (n=50). Lines are statistically significant linear regressions

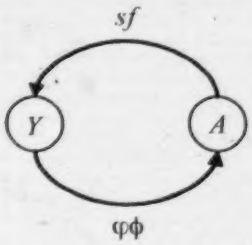

Figure 5.4: Life cycle graph for pink salmon.

salinity for any developmental stage (Figure 5.3).

C. clemensi followed a very different epizootiology. For the first three months of pink salmon marine life, *C. clemensi* was more abundant than *L. salmonis* ($p<0.001$ for April, May, and June for all years 2003-2005; Fig. 5.5). Note that there were no April data for 2006. Infection prevalence of *C. clemensi* was 8-20% during this time with a clear developmental progression resulting in many motile stage lice in June and July. The sustained rate of new infections (evidenced by copepodid and chalimus lice presence during April-June) plus the accumulation of motile lice resulted in a marked increase of *C. clemensi* in July ($p<0.001$). This contrasts with the more abrupt increase in *L. salmonis* in July. During July the dominance of *C. clemensi* diminished when *L. salmonis* abundance increased and, in 2005, exceeded *C. clemensi* ($p<0.001$). The abundance of *C. clemensi* copepodids and chalimus lice was significantly related to salinity (Figure 5.3).

5.5 Model and Analysis

The previous section suggests migratory allopatry protects juvenile pink salmon from *L. slamonis* but not *C. clemensi* in early marine life. In previous sections I have shown that salmon farms can undermine migratory allopatry by increasing *L. salmonis* exposure of wild juvenile salmon. To theoretically assess the importance of migratory allopatry for pink salmon population dynamics I investigated the effects of increasing *L. salmonis* exposure of juvenile salmon using an empirically parameterized stage-structured host population model with survival terms determined partly by parasites. The model decomposes juvenile salmon mortality into parasite (φ) and non-parasite (ϕ) associated terms, which have different formulations depending on whether or not predation is selective on infected fish. For pink salmon the juvenile stage (Y) corresponds to the first three months of marine life when they are allopatric with large abundances of adult (A) salmonids. The host lifecycle can be described by the simple graph (Figure 5.4) where f is the

number of juveniles produced per adult, s is the proportion of adults that survive to spawn. The net reproductive value, R_0, of the fish population (not the parasite) can be read directly from the graph as $R_0 = \varphi\phi sf$. At low population density it is of interest to know if $R_0 > 1$ which means that adults produce on average more than one adult offspring in their lifetime and the population will grow. As R_0 increases, so too does the resilience of the population – the population will recover faster after perturbation. Alternatively if $R_0 < 1$ individuals cannot on average replace themselves and the population will decline to extinction (Caswell, 2002).

To track changes in population abundance I introduce density dependence into reproduction through a modified Ricker equation $g = A\exp[r(1 - A/K)]$ (Ricker 1954) and replace f with g. The reproduction equation g accounts for competition for spawning habitat where female salmon can damage nests and eggs made by other females, a process known as redd superimposition (Heard 1991; Quinn 2005). Here, K is the carrying capacity of spawners and e^r is the number of juveniles produced per spawner at low density (the parameter r is $r = \ln(f)$). The equilibrium abundance of adult salmon is $A^* = [K/r][\ln(\varphi\phi s) + r]$ and for juveniles it is $Y^* = [K/(\varphi\phi r)][\ln(\varphi\phi s) + r]$. When sea lice abundance is at natural levels we denote the adult equilibrium abundance as $\tilde{A}^*$. To gauge the effect of increasing parasite exposure of juvenile salmon on wild salmon population abundance I define $\hat{A}^* = A^*/\tilde{A}^*$ which is the realized abundance relative to population abundance. At natural sea lice levels $\hat{A}^*$ equals 1 when there is no population depression and equals 0 when the population is extirpated.

The salmon population dynamics occur in discrete two-year cycles but parasite and non-parasite associated mortality occur in continuous time within host cohorts. To capture this I needed continuous time survival models that define φ and ϕ. The proportion of juvenile salmon surviving parasitism can be expressed by the survival model

$$\varphi = \exp\left[-\int_0^T \Phi(t)dt\right], \tag{5.1}$$

where $\Phi(t)$ is the rate of parasite induced host mortality of juvenile salmon, which can take different forms depending on the interaction between parasitism and predation. T is the duration of the juvenile stage (3 months). Let α be the host mortality rate of juvenile salmon induced per parasite and μ be the rate of non-parasite related mortality of juvenile salmon. If parasite induced host mortality is not compensatory (i.e. acts independently of other mortality factors) then the rate of parasite induced host mortality is simply

$$\Phi_1(t) = \alpha\overline{P}(t), \tag{5.2}$$

where $\overline{P}(t)$ is the mean parasite abundance on juvenile hosts at time t. Alternatively,

one could presume that infection induces compensatory mortality because predators, which are the primary presumed proximate cause of mortality (Groot and Margolis, 1991), may selectively prey on infected fish and thereby not change the actual number of fish killed. In this scenario the only additional mortality caused by infection will be from parasites surviving predation of their immediate host. The rate of parasite induced host mortality for this compensatory model is

$$\Phi_2(t) = \begin{cases} 0 & , \text{ if } \alpha\overline{P}(t) < \mu(t) \\ (\alpha\overline{P}(t) - \mu(t)) & , \text{ if } \alpha\overline{P}(t) > \mu(t) \end{cases} . \tag{5.3}$$

Note that equation 5.3 defines a parasite abundance threshold, $\bar{P}_c(t) = \alpha(t)/\mu(t)$, below which there are no population impacts.

I do not include explicit models for sea lice population dynamics on the juvenile salmon because the timescale of the sea lice lifecycle is slow (1.5 months) relative to the exposure period (1-3 months). This means that the sea lice dynamics on the juvenile salmon can be well approximated by an immigration and death process where sea lice abundance on juvenile salmon is controlled by transmission from natural and/or farm reservoir hosts and subsequent lice survival on juvenile salmon. Explicit inclusion of lice population dynamics on juvenile salmon could result in an increase through time (because of subsequent generation of lice) but this could be also balanced by declines in transmission from reservoir hosts the juvenile salmon have long passed during their migration. Because I am interested in the general sensitivity of salmon population dynamics to lice exposure I simply control lice abundance as an exogenous variable.

To parameterize the model I used data on pink salmon fecundity, freshwater and marine survival summarized in Heard (1991). Because there is considerable temporal and spatial variation in these parameters I tried to be conservative in the parameter estimates. I chose to be conservative so that any uncertainty in parameter values would minimize the impact of the sea lice on the salmon population dynamics. As shown below, the conservative estimates tend to over-estimate the reproductive capacity of the salmon R_0. Starting with reproduction, I assume that females carry, on average, 1600 eggs (Table 2 in Heard (1991)), and that fertilization success is 100%. To estimate egg to fry survival we needed to control for density dependence during spawning. To tease apart density dependence from abiotic factors affecting egg to fry survival we compared egg to fry survival estimates from natural rivers (mean = 10.8%) and spawning channels (mean = 50.3%) in British Columbia (see Table 17 in Heard (1991)). Assuming that density dependence and abiotic factors are roughly independent, that density dependence affects populations spawning in both natural streams and spawning channels, and that abiotic mortality factors are minimal in artificial channels, we calculated that the average density independent

egg to fry survival in natural streams is 10.8/50.3=0.21%. These calculations mean that at low spawner density each female produces 336 fry.

I estimated the subsequent survival from marine emergence through spawning using an established within cohort mortality schedule for pink salmon estimated from detailed long-term observations from a central British Columbia population (Figure 36 in Heard (1991)). The estimated mortality schedule must intrinsically account for the natural effects of parasitism. For early marine life of juvenile salmon, the instantaneous rate of mortality at natural parasite levels is $\nu(t) = 0.53$ month^{-1}. When mortality in non-compensatory then parasites have an additive effect which must be subtracted to estimate non-parasite associated mortality. When mortality in non-compensatory then parasites have an additive effect which must be subtracted to estimate non-parasite associated mortality $\mu_1(t) = \nu(t) - \alpha\bar{P}(t)$. From the previous chapter, I have estimated $\alpha = 0.69$ (motile lice·month)$^{-1}$ and from studies where there are no farms, I know that $\bar{P}(t) \approx 0.009$ motile lice and so we can calculte $\phi_1 = \exp[-\int \mu(t)dt] = 0.21$. When mortality is compensatory, $\mu_2(t) = \nu(t)$ (parasites are removed by predators and have no additional effect), and we can calculate $\phi_2 = \exp[-\int \mu_2(t)dt] = 0.20$. The survival of salmon through the remainder of their lifecycle was calculated as $s = \exp[-\int \gamma(t)dt] = 0.25$ where $\gamma(t)$ is the instantaneous mortality rate of pink salmon from the third month of marine life through spawning (Figure 36 in Heard (1991)). These calculations mean that approximately 5% of pink salmon fry will return as adults under natural parasite abundances. For our purposes we chose a carrying capacity of 100,000 adults.

With these parameter values, I explored the consequences of compensatory interactions between predation and parasitism and increasing parasite exposure (abundance of motile lice per fish and temporal duration of exposure) on juvenile salmon survival, salmon population persistence (via the salmon net reproductive value R_0) and the remaining salmon population abundance relative to abundance at natural sea lice levels. I found that pink salmon populations are highly sensitive to parasitism of juvenile fish, but that compensatory mortality creates a threshold value of $\bar{P}_c(t) = \alpha(t)/\mu(t) = 0.75$ motile lice per fish below which there are no effects on salmon population dynamics. This threshold value places a theoretical upper bound on the mean abundance of sea lice per juvenile fish, below which there are no population impacts. The true value would probably occur between zero and $\bar{P}_c(t)$, depending on the exact nature of the interaction between parasites and predators. As the parasite abundance increased, there were initial sharp declines in wild salmon abundance and resilience (Figure 5.5). Full population collapse occurred when $R_0 < 1$, meaning that when lice abundances causing $R_0 < 1$ are sustained over several salmon generations the salmon populations may be extirpated. In the most

conservative case - compensatory mortality and only a one-month period of sea lice exposure - population collapse was predicted at a mean infection abundance of approximately five motile lice per fish. At the other extreme non-compensatory mortality and three months of exposure - population collapse was predicted to occur at a mean louse abundance of approximately 1.5 motile lice per fish. These results are conservative based on the value of $R_0 \sim 17$ at zero lice abundnce which is high relative to the value of 3.3 estimated from stock-recruit data (Myers et al., 1999).

5.6 Discussion

These results suggest migratory allopatry protects juvenile pink salmon from *L. salmonis* in early marine life. Because salmonids bound the host range of *L. salmonis* the parasites life history is dominated by its hosts migration. The bulk of adult salmon populations are offshore and beginning their return migration when juveniles are in coastal waters and beginning their ocean migration (Groot and Margolis, 1991; Quinn, 2004). This explains the low *L. salmonis* prevalence (2-3 %) during the first 2-3 months of pink salmon marine life. These lice probably originated from local small populations of cutthroat trout (*O. clarki*) and dolly varden (*Salvelinus malma*) and scattered coastal ocean rearing chinook that are orders of magnitude less abundant than other Pacific salmonids with oceanic migrations. Overall, *L. salmonis* did not reach appreciable numbers until the return migration of adult Chinook salmon in July when abundances of all louse developmental stages abruptly increased. This suggests cross-generational transmission occurs both through infectious larvae and direct transmission of motile lice, which can move among host fish (Ritchie, 1997). It is important to note that during the *L. salmonis* refuge juvenile pink salmon are highly vulnerable to *L. salmonis* infection: they lack scales when young and weigh 0.5-2 g, which is three or four orders of magnitude smaller than adults. Survival of juvenile pink salmon infected with one or two motile *L. salmonis* is low (Morton and Routledge, 2005; Chapter 4). Taken together these data suggest that migration causes host-age structuring with first a freshwater/marine transmission barrier and then an allopatric marine transmission barrier that define a refuge from *L. salmonis* when the host is ontogenetically most vulnerable to infection.

In contrast, *C. clemensi* followed a different epizootiology explained mostly by host diversity. The host range of *C. clemensi* includes locally ubiquitous populations of herring, abundant sandfish, and scattered stickleback that are sympatric with juvenile salmon. Correspondingly, juvenile salmon encountered *C. clemensi* shortly after marine emergence and infections were sustained at a level of 8-20%. Further I did not detect an increase in *C. clemensi* infection that could be attributed

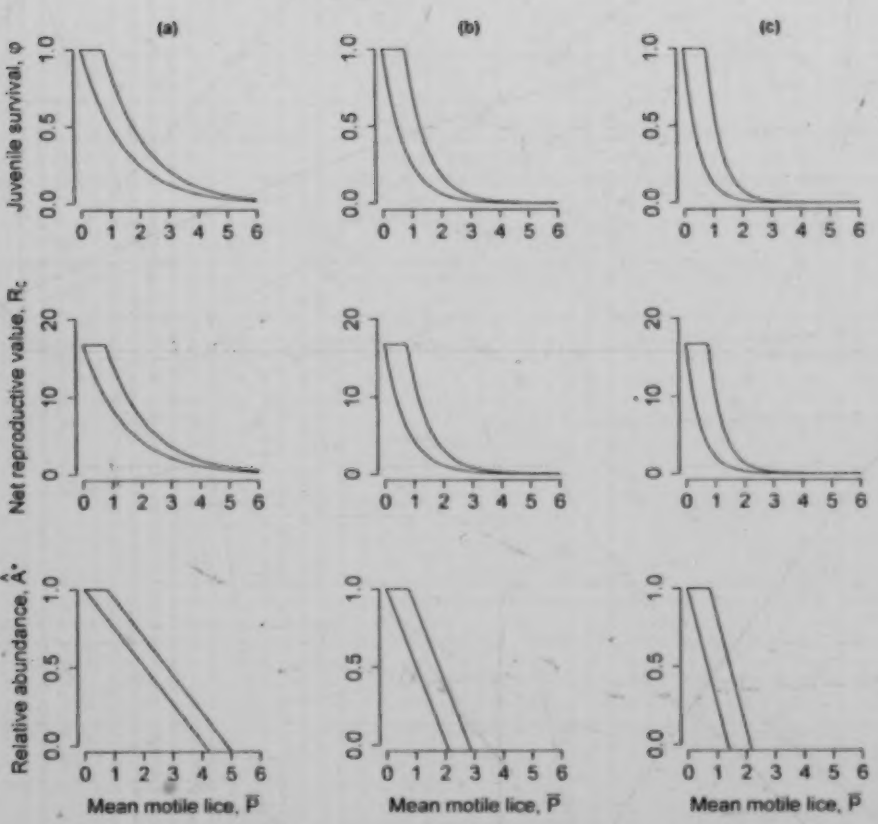

Figure 5.5: Effects of increasing motile sea lice infection, $\overline{P}$, of juvenile pink salmon on juvenile salmon survival (φ), salmon net reproductive value (R_0), and salmon abundance relative to abundance at natural lice levels ($\hat{A}^*$) for (a) one month exposure, (b) two months exposure, and (c) three months exposure. Regions bound the predictions between compensatory (right boundary; equation 5.3) and non-compensatory (left boundary; equation 5.2) parasite induced host mortality. The horizontal dotted line shows R_0=1, above which salmon populations persist and below which salmon populations collapse.

to the return of adult salmon – the rate of new infections remained the same during this time (Table 5.1) and we observed only one gravid *C. clemensi* from among the thousands of lice on the adult Chinook salmon that we opportunistically sampled. Gravid *C. clemensi* on returning adult Pacific salmon have also been rare in other field surveys (Beamish et al., 2005). These findings suggest *C. clemensi* infections are not sustained over the off-shore portion of the salmon lifecycle and that the parasite is primarily distributed in coastal ecosystems. Possible explanations include: *C. clemensi* may not be successfully transmitted in pelagic environments, *L. salmonis* may competitively exclude *C. clemensi*, or salmon are generally incompetent hosts sustaining *C. clemensi* through immigration from source host populations extant in nearshore but not pelagic ecosystems (the rescue effect in source-sink dynamics)(Pulliam, 1988). It is also important to note that *C. clemensi* is smaller and apparently less pathogenic than *L. salmonis* to juvenile salmon. We did not observe mechanical damage to surface tissues of infected fish or other signs of pathology that are associated with *L. salmonis* infection (Morton and Routledge, 2005). Taken together these findings suggest that host diversity maintains *C. clemensi* infections of juvenile pink salmon but that infection is only an ephemeral feature of pink salmon life history.

Increasing *L. salmonis* exposure of wild juvenile pink salmon may threaten wild pink salmon populations. Pink salmon populations have collapsed following *L. salmonis* epizootics (PFRCC, 2002) and rebounded after fallowing migration routes (Morton et al., 2005; Beamish et al., 2006). The model results explain these patterns and predict a significant disease threat of aquaculture to pink salmon populations. Infections of 1-3 motile *L. salmonis* per fish for 1-3 months are predicted to result in salmon population collapse whether or not mortality from sea lice is compensated by selective predation. The model also predicts a more subtle effect – rapid loss of population resilience. Populations exposed to mean parasite abundances of less than one motile louse per fish rapidly lose their ability to recover from perturbation. The model is a simple combination of established fisheries and host-parasite models that were parameterized with long established (Groot and Margolis, 1991) and extensive (Chapter 4) datasets. Nevertheless, the model did not account for other factors such as climate or ocean circulation changes that could dampen or amplify the predictions. Model predictions could also be improved with more detailed survival data of infected juvenile salmon over a range of body masses. Despite these limitations, it is clear that migratory allopatry naturally protects wild juvenile pink salmon from *L. salmonis* and that pink salmon populations are sensitive to breaking this transmission barrier and increasing *L. salmonis* exposure of wild juvenile salmon. In the next chapter I turn to data on the number of

pink salmon returning annually to rivers on the central coast of British Columbia to empirically test for the effect of *L. salmonis* infestations on wild pink salmon population dynamics.

Spp/n	Year	Stage	April	May	June	July
n	2004		415	2295	2637	893
n	2005		3323	4383	3549	990
n	2006		no data	1028	1035	900
Lep	2004	C	0	0	0.0008 (0, 0.002)	0.0011 (0, 0.0034)
		H	0.0024 (0,0.0072)	0.0096 (0.006, 0.014)	0.004 (0.002, 0.006)	0.0056 (0,0.011)
		M	0	0.0026 (0.00087, 0.0050)	0.0099 (0.0064, 0.014)	0.10 (0.080, 0.13)
	2005	C	0.0012 (0.00030, 0.0024)	0.0023 (0.00091, 0.0039)	0.0019 (0.00056, 0.0036)	0.20 (0.17, 0.23)
		H	0.0018 (0.00060, 0.0033)	0.013 (0.0093,0.016)	0.0045 (0.0023, 0.0067)	0.098 (0.073, 0.13)
		M	0	0.0011 (0.00023, 0.0023)	0.020 (0.015, 0.025)	0.21 (0.17, 0.24)
	2006	C	-	0.0039 (0.00097, 0.0078)	0.011 (0.0048, 0.018)	0.02 (0.012, 0.030)
		H	-	0.0029 (0, 0.0068)	0.00097 (0, 0.0030)	0.011 (0, 0.033)
		M	-	0	0.035 (0.023, 0.046)	0.072 (0.056, 0.091)
Cal	2004	C	0.029 (0.014, 0.046)	0.013 (0.0087, 0.017)	0.024 (0.018, 0.030)	0.0056 (0.0011, 0.010)
		H	0.031 (0.014, 0.048)	0.057 (0.037, 0.058)	0.071 (0.059, 0.084)	0.060 (0.034, 0.096)
		M	-	0.0013 (0, 0.0031)	0.013 (0.0087, 0.019)	0.12 (0.096, 0.15)
	2005	C	0.060 (0.051,0.069)	0.031 (0.026, 0.037)	0.0054 (0.0028, 0.0079)	0.022 (0.013, 0.031)
		H	0.039 (0.032, 0.046)	0.15 (0.13, 0.16)	0.046 (0.038, 0.054)	0.20 (0.15, 0.24)
		M	0	0.0016 (0.00046, 0.0027)	0.013 (0.0099, 0.017)	0.19 (0.15, 0.23)
	2006	C	-	0.024 (0.016, 0.034)	0.019 (0.012, 0.029)	0.077 (0.059, 0.096)
		H	-	0.032 (0.021, 0.044)	0.0039 (0.00097, 0.0077)	0.020 (0.011, 0.031)
		M	-	0.0029 (0, 0.0068)	0.11 (0.080, 0.14)	0.20 (0.17, 0.23)

Table 5.1: Total sample sizes (n) and mean abundances of sea lice developmental stages (± 95% bootstrap confidence intervals) for each sampling period for *L. salmonis* (Lep) and *C. clemensi* (Cal) for years 2004-2006. Sea lice stages are divided into copepodid/chalimus I (C), chalimus II-IV (H), and motiles (M).

Chapter 6

Salmon population dynamics in relation to louse infestations*

6.1 Introduction

In previous chapters I have shown that juvenile salmon are naturally protected from *L. salmonis* by migratory allopatry, that lice can spread from farm salmon to wild juvenile salmon, that lice from farms can cause epizootic mortality in wild juvenile slamon, and that wild salmon populations are theoretically sensitive to louse exposure of juvenile fish. I have not yet empirically tested for an effect of sea lice infestation on wild salmon populations. Sea lice have infested wild juvenile pink salmon in the Broughton Archipelago in 2001 (Morton and Williams, 2003) and 2002 (Morton et al., 2004) as well as 2003-2005 (Chapters 3-4, Morton et al. 2005). There is no evidence of significant infestations prior to 2001, and given that the Broughton Archipelago is inhabited by First Nations people, biologists, and fishermen, it is doubtful these would have gone unnoticed. From these five years of infestations, 2001-2005, one can evaluate the effects of sea lice on wild pink salmon population dynamics. In this chapter, I statistically test for and quantify the effects of sea lice on wild pink salmon populations using a model that connects data on sea lice infestations and data on wild salmon population dynamics.

6.2 Data

The salmon data consist of Fisheries and Oceans Canada escapement data (the number of salmon per river) from 1970 to present for all pink salmon populations from rivers in the central coast of British Columbia, Canada (Figure 6.1). There were 64 rivers not exposed to salmon farms and 7 rivers whose salmon populations

*The methods and results of this chapter have been published in Krkošek, M., Ford, J.S., Morton, M, Lele, S., Myers, R.A., and Lewis, M.A., 2007. Declining wild salmon populations in relation to parasites from farm salmon. *Science*, 318, 1772-1775.

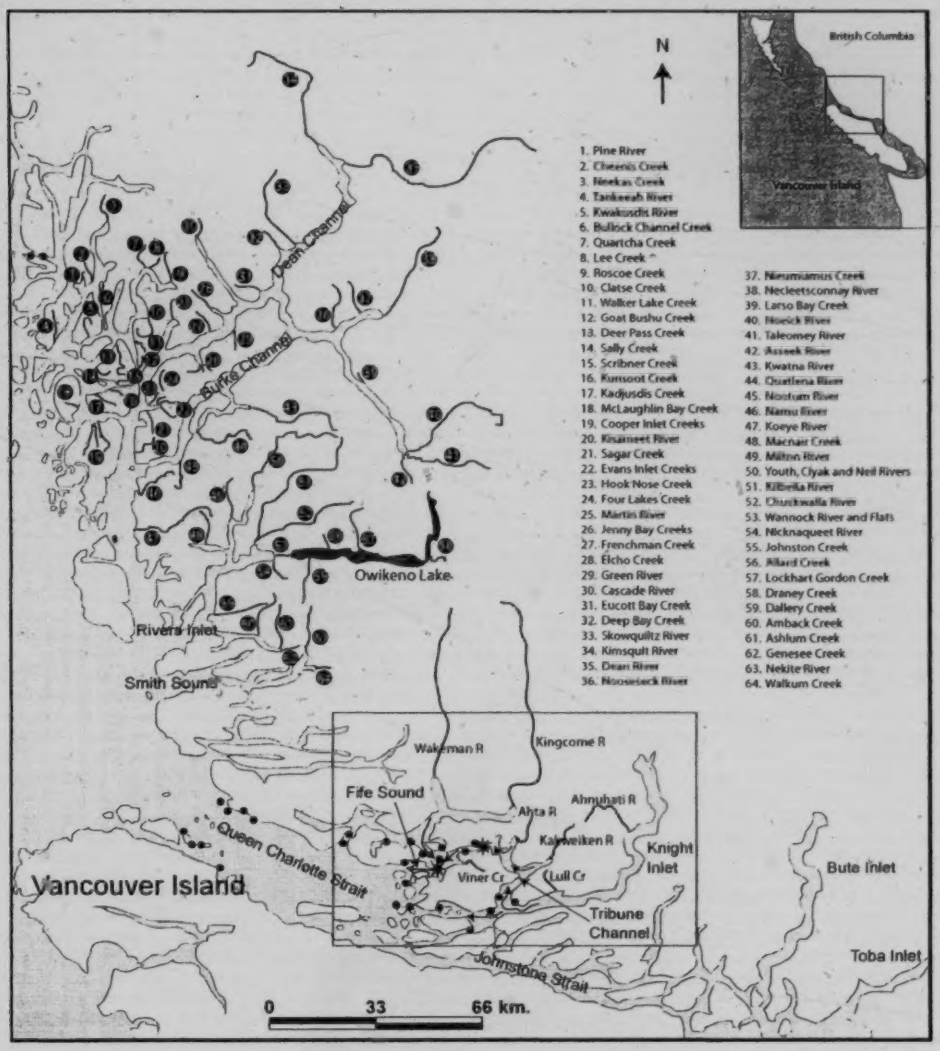

Figure 6.1: Study area depicting pink salmon populations from control areas (numbered) and exposed areas (directly labeled) in the Broughton Archipelago (boxed area). Inferred migration routes in the Broughton Archipelago are shown by arrows. Salmon farms are shown by black dots and sample sites by stars. The eastern sample site is known as Glacier Falls and the western sample site is known as Burdwood Islands. The fallowed migration route in 2003 consists of Tribune Channel through Fife Sound but farms peripheral to this route remained active. Salmon farms south of Knight Inlet are not shown.

must migrate past at least one salmon farm. Because pink salmon have a two-year lifecycle there are distinct odd and even year lineages (Heard, 1991), which amounts to 128 unexposed populations and 14 exposed populations. Rivers with substantial enhancement (e.g. spawning channels) were excluded because any increased abundances in these rivers confound estimates of natural changes in abundance. Unexposed populations had been and continue to be commercially fished. Exposed populations were commercially fished before the infestations but the fishery remains closed since the onset of the infestations.

The louse data consist of weekly collections of 30-50 juvenile pink salmon in the spring and early summer from two sites (Figure 6.1). The Glacier Falls site represents populations from the Ahta and Kakweiken rivers. The Burdwood Islands site represents populations from the Wakeman, Kingcome, and Viner rivers. We used a modified infection index for populations from the Lull and Ahnuhati rivers that assumes half of the populations migrate through Tribune Channel and the other half migrate out Knight Inlet where louse abundance is about half that in Tribune Channel (because there are fewer salmon farms and greater potential for the dispersion of infectious larvae in Knight Inlet). This last assumption can be verified from the intensive surveys I conducted on these migration routes in 2003 and 2004 (Chapters 3-4). The data for the populations from the seven rivers were therefore organized into three groups: Glacier Falls, Burdwood Islands, and Knight Inlet. Because estimates of louse abundance for the Knight Inlet group are not indirect, I analyzed the data with and without the Knight Inlet group included. Further, because the estimates of louse abundance were not independent for populations within each group, I also conducted the same analysis with the abundance data averaged (within each year) over populations corresponding to the Burdwood Islands, Glacier Falls, and Knight Inlet sample sites. This reduced the sample size from 35 to 10 data points for model fitting.

6.3 Modeling pink salmon population dynamics

I used a classical stock-recruit relation, known as the Ricker equation (Ricker, 1954), to model the pink salmon population dynamics. The deterministic model is

$$n_i(t) = n_i(t-2)\exp[r - bn_i(t-2)] \tag{6.1}$$

where $n_i(t)$ is the scaled abundance of population i in year t, r is the population growth rate, and b determines density dependent mortality. The scaled abundance is $n_i(t) = N_i(t)/m_i$, where $N_i(t)$ is the escapement estimate for population i in year t and m_i is the average escapement (from 1970 to 2006) for population i. This scaling was done to remove variation among populations due differences in the availability

Dataset	Random effects	Parameters	L	BIC	ΔBIC
Unexposed	r and b	4	2642.92	5329.81	18.19
	b	3	2642.93	5315.16	3.54
	r	3	2644.82	5318.95	7.33
	None	2	2644.82	5311.62	0
Exposed pre-infestaion	r and b	4	334.23	699.8	15.7
	b	3	334.23	689.4	5.2
	r	3	334.23	689.4	5.2
	None	2	334.23	684.2	0

Table 6.1: Comparison of hierarchical and non-hierarchical Ricker models in their fits to escapement data from unexposed and exposed pre-infestation populations, showing the number of parameters, negative log likelihood (L) values, Bayesian Information Criteria (BIC), and the difference in BIC values between each model and the best fit model.

of suitable habitat and so make all populations comparable. Pink salmon are known for high variation in population abundance due to climatic and other random factors. Similar to other studies (Myers et al., 1999), I represent this stochasticity with a lognormally disributed random factor

$$n_i(t) = n_i(t-2)\exp[r - bn_i(t-2)]\exp[N(0,v)], \tag{6.2}$$

where N is a random variable from a normal distribution with mean zero and variance v. Upon log transformation to

$$\log[n_i(t)/n_i(t-2)] = r - bn_i(t-2)] + N(0,v) \tag{6.3}$$

the Ricker equation becomes a linear model with intercept r and slope b that can be estimated using linear regression. The 95% confidence intervals on r and b must be estimated using parametric bootstrapping (Dennis and Taper, 1994). When analyzing multiple time-series from different populations it may be necessary to use heirarchical mixed effects models that represent variation among populations in the deterministic parameters r and b (Myers et al., 1999; Pinheiro and Bates, 2004). The equation for the hierarchical mixed effects model includes random effects on both r and b:

$$\log[n_i(t)/n_i(t-2)] = (r + \mu_r) - (b + \mu_b)n_i(t-2) + N(0,v) \tag{6.4}$$

where μ_r and μ_b are normally distributed over the populations with mean zero and variances to be estimated.

To select the best model for pink salmon population dynamics I first analyzed unexposed and exposed pre-infestation datasets which have sufficient data to support heirarchical mixed modeling (long time series from each population). I compared

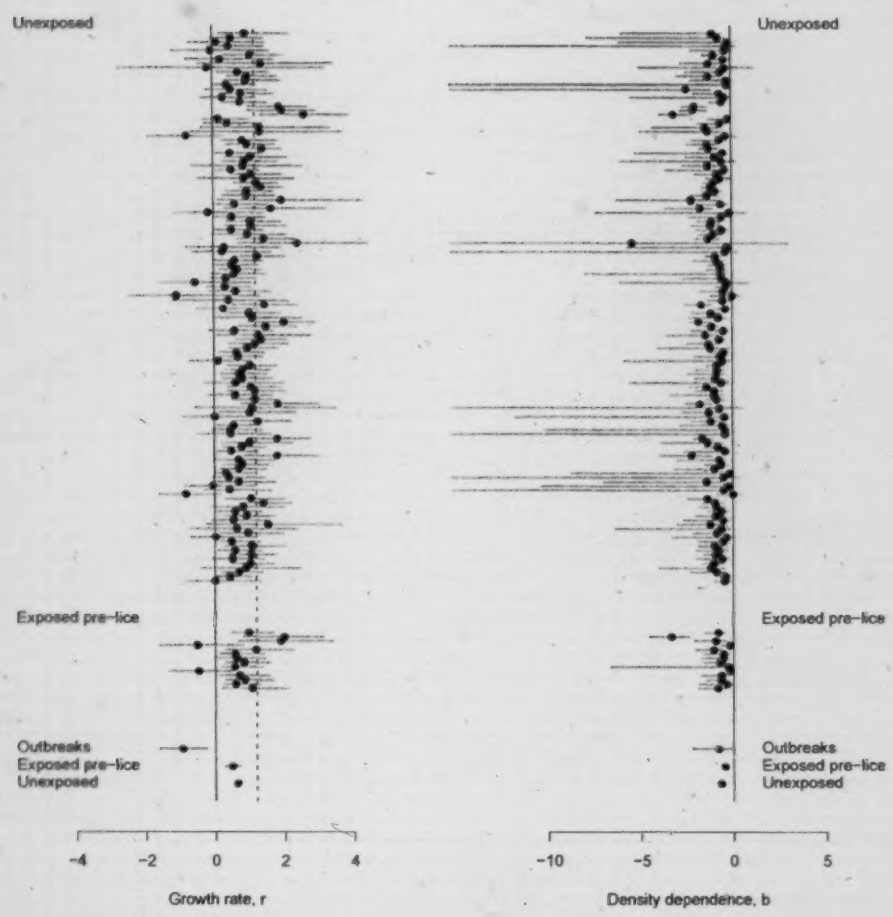

Figure 6.2: Estimates (dots) and 95% confidence intervals (grey lines) for growth rate (r) and density dependence (b) in the Ricker equation when fit independently to each of the 128 unexposed populations and the 14 exposed populations prior to infestations. Also shown are estimates and 95% confidence intervals for r and b in the Ricker equation when fit to grouped data: unexposed populations, exposed populations prior to the infestations, and exposed populations during the infestations. The 95% confidence intervals were calculated by parametric bootstrapping (Dennis and Taper, 1994). The vertical dotted line corresponds to the maximum reproductive value, r^*=1.2, calculated for pink salmon (Myers et al., 1999).

models with and without random effects on r and b using Bayesian Information Criteria (BIC) for model selection inference. There was little variation among populations in the estimates for r and b (Figure 6.2), and statistically, the data did not support including random effects in the model (Table 6.1). This means the data from multiple populations can be pooled and the heirarchical structure of the model can be dropped (Pinheiro and Bates, 2004), leaving the stochastic Ricker model model (equation 6.2) to be fit to the pooled data using simple linear regression to estimate parameters (Pinheiro and Bates, 2004) and parametric bootstrapping to estimate the 95% confidence intervals on the estimated parameters (Dennis and Taper, 1994).

6.4 Effects of lice on pink salmon population dynamics

6.4.1 Pink salmon population growth rates

Prior to the sea lice infestations that began in 2001, Broughton Archipelago pink salmon populations fluctuated in a manner similar to unexposed populations, but during the infestations there is a sharp decline in their productivity (Figures 6.3-6.4). I compared parameter estimates of the Ricker model among three groups: unexposed populations, pre-infestation exposed populations, and exposed populations during infestations (excluding the fallow year). The groups did not differ in b and so I reanalyzed the data with b fixed among the three groups. Unexposed populations did not differ from exposed pre-infestation populations in growth rate (unexposed populations: r = 0.62, 95% CI: 0.56 to 0.70; exposed pre-infestation populations: r = 0.70, 95% CI: 0.47 to 0.92). The growth rate of exposed populations during the infestations was significantly lower and significantly negative (r = -1.17, 95% CI: -1.68 to -0.62); Figure 6.5). I initially excluded the fallow data because they contain only one year and correspond to a non-random management action. By fixing b=0.64, as estimated above, and estimating r from the remaining seven data points I found the growth rate of fallow populations was significantly increased (r = 2.63, 95% CI: 1.39 to 3.78). The maximum reproductive rate for pink salmon is r^* = 1.2 (Myers et al., 1999). Fishing mortality probably reduced r for unexposed and exposed pre-infestation populations. The depressed growth rate of exposed populations during the infestations indicates that previous fishing mortality (now ceased) has been greatly exceeded by lice. These effects of sea lice and fishing on pink salmon population dynamics is summarized in Figure 6.6.

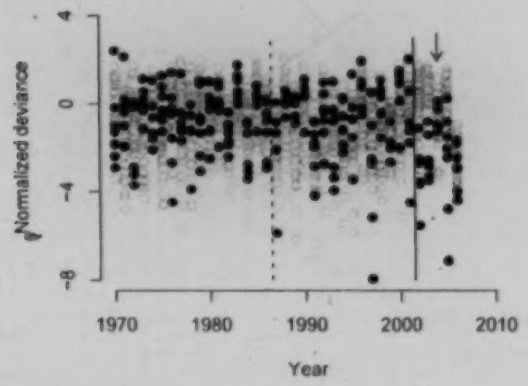

Figure 6.3: Time series of normalized population deviances ($\log[N_i(t)/m_i]$, where $N_i(t)$ is the population estimate for population i in year t and m_i is the time series mean abundance for population i) for 128 control populations (light grey open circles) and 14 Broughton Archipelago pink salmon populations exposed to salmon farms (black dots). The vertical dotted line marks the beginning of salmon aquaculture in the Broughton Archipelago. The vertical solid line marks the onset of louse infestations (and the commercial fishery closure) affecting the exposed populations. The arrow indicates data for exposed pink salmon cohorts that, as juveniles, experienced a fallowed migration corridor.

6.4.2 Pink salmon population viability analysis

The population growth rate of Broughton pink salmon was significantly negative during infestation years (Figure 6.6). According to the theory of population viability analysis, this means extinction of Broughton pink salmon populations is certain if sea lice infestations continue (Dennis et al., 1991). I followed the methods in Dennis et al. (1991) to estimate the mean time to extinction for Broughton pink salmon, should the infestations continue. Dennis et al. (1991) begin with a simple linear model of exponential population growth or decay. They assume, based on other analytical and simulation studies, that the exponential model, when log-tranformed and coupled with environmental stochasticity, is well-approximated by a Wiener process with drift. They define $X(t) = \log[n(t)]$, where $X(t)$ is the log transformed population abundance and is normally distributed (with mean $x_0 + \mu t$ and variance $\sigma^2 t$). For their model, μ is the population growth rate and is estimated from the fit of the exponential model to time-series data (simply the mean of $\log[n(t)/n(t-\tau)]$ when population abundance is estimated for each generation and τ is the generation time). Environmental stochasticity, σ^2, is estimated as the variance of residuals from the fit of the exponential model (the residuals of the data from the predicted abundance $x_0 + \mu t$). For my purposes, this estimation of μ and σ^2 is not valid

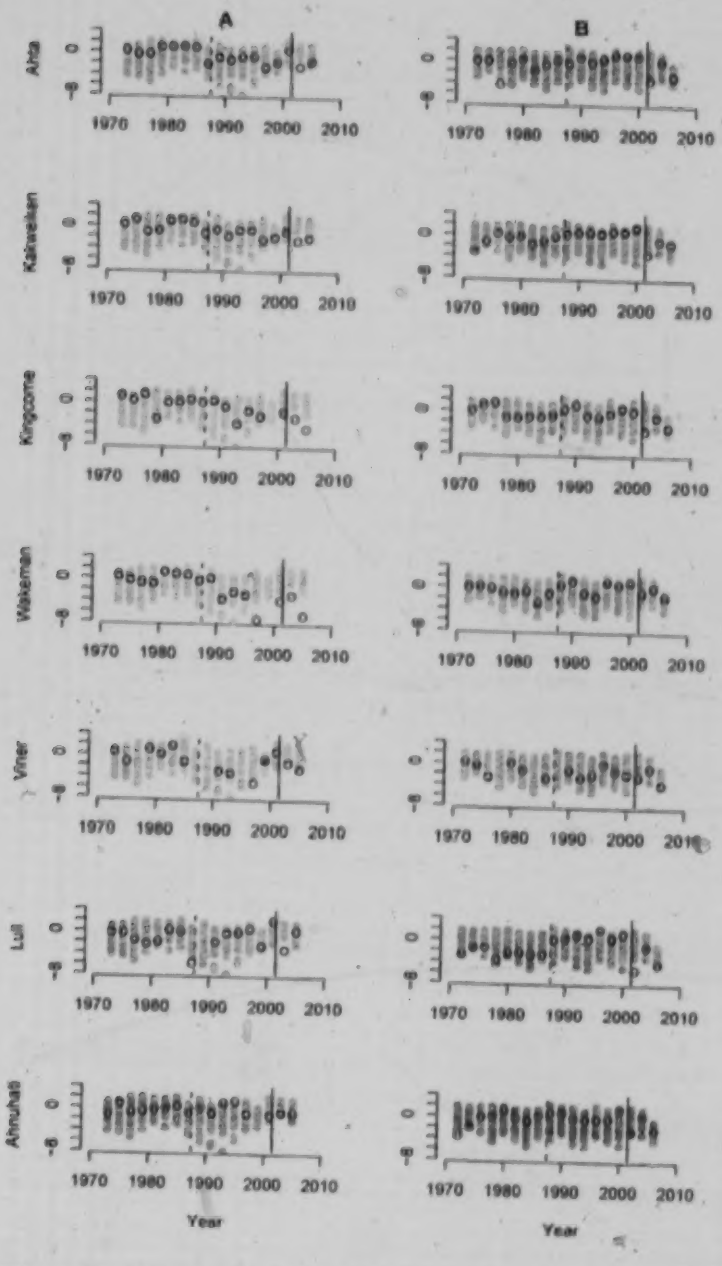

Figure 6.4: Normalized time series of pink salmon escapements to each river in the Broughton Archipelago (black dots) and unexposed rivers (grey dots) for (A) odd year lineages and (B) even year lineages. The data were normalized by dividing escapement estimates by the time-series mean for each population (to make all populations comparable) and then taking the natural logarithm.

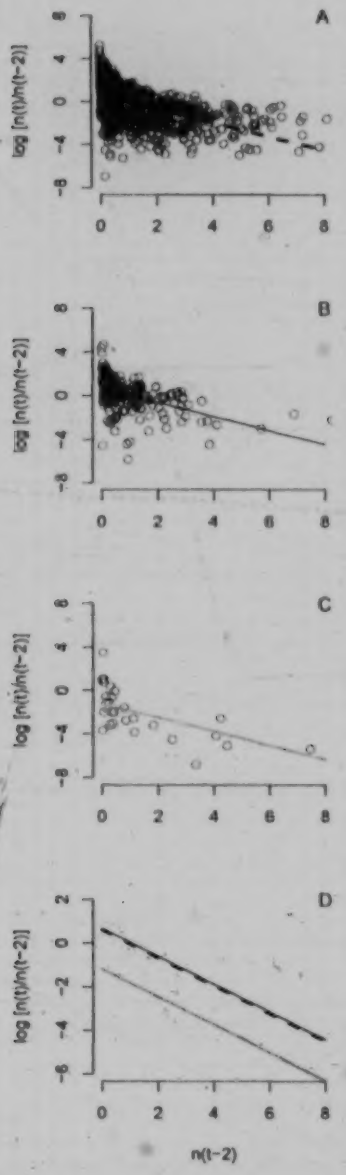

Figure 6.5: Fits of the log transformed Ricker model to escapement data for A, unexposed populations, B, exposed populations prior to infestations, C, exposed populations during the infestations, and D, a comparison of the log transformed Ricker model for the three groups in panels A-C. The intercept (growth rate) is lower for the exposed population during the infestations than for exposed populations before the infestations and the unexposed populations.

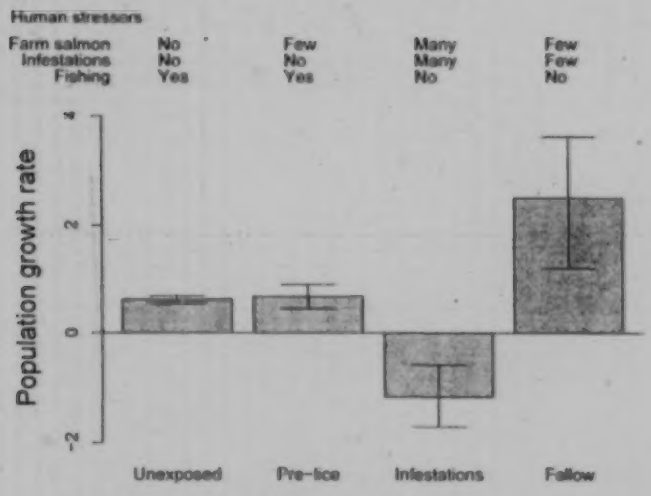

Figure 6.6: Effects of fishing, aquaculture, and sea lice infestations on the population growth rate, r of pink salmon populations in the Broughton Archipelago.

because density dependence is present in the data (Figure 6.2). Instead, I estimated the population growth rate by fitting the log-tranformed Ricker model and calculated the variance due to environmental stochasticity from the residuals of the fit of the log transformed Ricker model. As population abundances decline, density dependent declines in survival or reproduction due to competition should become negligible and the Ricker model then converges to the exponential model. Thus, I am able to parameterize the exponential model from the fit of the Ricker model. The value of μ is simply the population growth rate (intercept of the log-transformed Ricker model) of the infestation group and environmental stochasticity, σ^2, was estimated from the residuals of the log-transformed Ricker model. Note that these estimates were taken from the fit of the log-transformed Ricker model to all escapement data (excluding fallow data) with b constrained to take the same value for all groups and r estimated separately for unexposed populations, exposed populations prior to the infestations, and exposed populations during the infestations. Defining population collapse as a 99% decline in population abundance, which has a value $n_e = 0.01$, I set $x_d = \log[1 - n_e]$, which is the log-transformed distance in population abundance from historical mean abundance to population collapse. I then used equations 91-93 in Dennis et al. (1991) to calculate the mean (and 95% confidence intervals) time to population collapse, which was 3.9 generations (95% CI: 3.7 to 4.2). During two generations of infestations some exposed populations have declined to $< 1\%$ while

Constraint	a	t	df	p
All populations included	-0.90	-5.323	34	6.55e-06
Knight Inlet excluded	-0.89	-5.617	24	8.80e-06
River groups averaged	-0.98	-3.716	14	0.0023
River groups averaged and Knight Inlet excluded	-0.94	-3.935	9	0.0034

Table 6.2: Results of t tests, over a range of data constraints, for including louse induced mortality of pink salmon in the Ricker model.

others have exceeded their historical abundance. The average abundance after two generations of infestations is 10.5%, which corresponds to 9.6% predicted by the model.

6.4.3 Pink salmon mortality estimates

To estimate the mortality of pink salmon caused by lice I extended the Ricker model to directly accommodate louse data collected from exposed populations during the infestations (Morton and Williams, 2003; Morton et al., 2004, 2005). I constrained the model by fixing $b = 0.64$ and by requiring $r = r^* = 1.2$ because there was no fishing mortality. Louse induced mortality is represented by extending the Ricker model to include parasitism

$$n_i(t) = n_i(t-2)\exp[r - bn_i(t-2)]\exp[-aP_i(t-1)]\exp[N(0,v)]. \qquad (6.5)$$

where P is the mean abundance of motile (adult and pre-adult) lice per juvenile salmon from population i that spawned in year t and is lagged one year to represent the fact that juvenile pink salmon migrate to sea one year before they return to spawn. I log transformed the model to

$$\log[n_i(t)/n_i(t-2)] = r - bn_i(t-2)] - aP_i(t-1)] + N(0,v). \qquad (6.6)$$

and so used linear regression to estimate a. The term $\exp[-aP_i(t-1)]$ significantly improved the fit of the model (t=-5.019, df=33, p=1.74e-05; Figure 6.7) and results remained strong when the data were restricted by averaging populations and excluding some population groups (all $p < 0.005$, Table 6.2).

Uncertainty in a can emerge from two sources: uncertainty in values of the constrained parameters, r and b, as well as uncertainty in the value of a from the fit of the model to the escapement and louse data. I observed little variation in the value of b among populations and so did not consider this source of uncertainty. To represent the remaining uncertainty in a, I implemented a hierarchical simulation where r was randomly drawn 1000 times from the known distribution of r^* (a normal distribution with mean 1.22 and variance 0.12) (Myers et al., 1999) and then 1000 values of a were drawn from the posterior distribution of a given r using

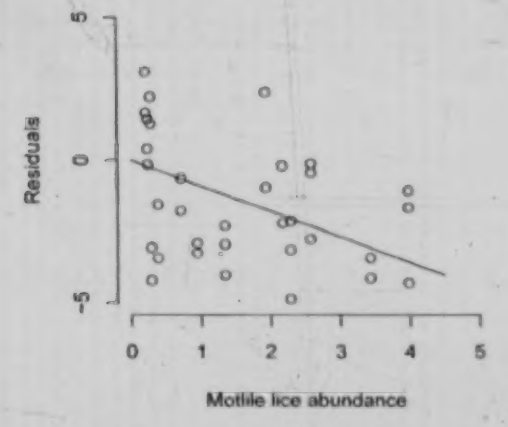

Figure 6.7: Residuals of the fit of the Ricker model with parameters constrained to $r = r^* = 1.2$ and $b = 0.64$ versus the corresponding motile louse abundance on juvenile salmon on exposed populations during infestation and fallow years. The solid line is the estimated impact of motile lice on salmon productivity with slope -0.89 (corresponding to a=0.89) and constrained to pass through the origin.

a uniform prior distribution of a on the interval (0,5). This resulted in a distribution of 1,000,000 estimates of a from which I calculated the 95% credible intervals.

The parameter a corresponds to the rate of parasite induced host mortality multiplied by the time juvenile salmon are exposed to the parasites, $a = \alpha T$. From the previous chapter I know the exposure time, T, is about 2 months (based on the migration speed of juvenile pink salmon through the archipelago) and the value of α is 0.022 (motile lice x day)$^{-1}$ (based on survival experiments of naturally infected juvenile pink salmon). Dividing the estimated $a = 0.90$ (95% credible intervals are 0.47-1.34) by 60 days reveals an excellent correspondence between these two independent estimates of pathogenicity ($a/60 = 0.015$, (95% credible estimates 0.0078-0.022)). The estimated mortality of pink salmon, $1-\exp[-aP_i(t-1)]$, caused by lice ranged from 16% to over 97% and was commonly over 80% (Table 6.3). The lowest mortality comes from fallow populations when louse abundance was nevertheless elevated possibly due to transmission from active farms outside the fallowed corridor. The parameter estimates further mean that the mean motile *L. salmonis* abundance on juvenile pink salmon differentiating population persistence and extinction is $r^*/a = 1.35$.

6.5 Discussion

These results suggest salmon farm-induced *L. salmonis* infestations of juvenile pink salmon have had a large negative effect on wild pink salmon populations in the Broughton Archipelago. Although there are many factors that drive fluctuations in pink salmon population dynamics, I carefully controlled for these other factors and isolated the effect of sea lice by using a comparative analysis. I studied pink salmon populations in a large region of British Columbia containing groups of populations that are exposed and unexposed to salmon farms. It is already known that all the populations fluctuate in synchrony (Pyper et al., 2001). In the analysis I first compared the unexposed and exposed populations before the sea lice outbreaks and the productivity of these groups were nearly identical. Then when sea lice infestations occurred for the exposed group, the productivity of the exposed populations declined sharply whereas it remained unchanged in the unexposed group. The increased growth rate in response to fallowing rules out other factors that could have affected exposed but not unexposed populations. Since all other factors except sea lice infestations are common to both exposed and unexposed populations, the natural conclusion is that sea lice caused the difference between the populations.

The results indicate sea lice infestations of Broughton Archipelago wild juvenile pink salmon have put the wild pink salmon in rapid decline and local extinction is immanent. The rate of decline for the Broughton Archipelago pink salmon populations during the sea lice infestations was rapid. The mean time for an average pink salmon population to transition from historical abundance to 99% collapse is four generations, under sea lice infestation conditions similar to 2001-2005. The Broughton pink salmon have been declining for two generations of sea lice infestations and are currently at 9.6% of their historical abundance. The model predicts the populations would be at 10.5% of their historical abundance after two generations of decline. The calculations suggest that during the infestations, frequently more than 80% of the annual pink salmon returning to Broughton Archipelago rivers were missing because they were killed by sea lice when they migrated out to sea. The estimated motile louse abundance threshold differentiating pink salmon population extinction and persistence is $r^*/a = 1.3$ motile lice per juvenile pink salmon. The results rely on extensive spatial replication to compensate for short time-series in infestation years. However, there is conservation risk associated with waiting for larger datasets because the estimated time to extinction is short.

River	2002		2003		2004		2005		2006	
	P	*M*	*P*	*M*	*P*	*M*	*P*	*M*	*P*	*M*
Ahta	3.4	95.21 (79.07,98.95)	1.0	59.09 (36.87,73.82)	0.3	23.82 (12.89,33.10)	2.6	90.21 (69.76,96.93)	0.4	30.06 (16.81,41.49)
Kakweiken	3.4	95.21 (79.07,98.95)	1.0	59.09 (36.87,73.82)	0.3	23.52 (12.89,33.10)	2.6	90.21 (69.76,96.93)	0.4	30.06 (16.81,41.49)
Viner	4.0	97.20 (84.12,99.53)	2.2	86.00 (63.65,94.76)	0.2	16.37 (8.79,23.51)	2.3	87.20 (65.29,95.41)	1.4	71.39 (47.48,84.68)
Wakeman	4.0	97.20 (84.12,99.53)	2.2	86.00 (63.65,94.76)	0.2	16.37 (8.79,23.51)	2.3	87.20 (65.29,95.41)	1.4	71.39 (47.48,84.68)
Kingcome	4.0	97.20 (84.12,99.53)	2.2	86.00 (63.65,94.76)	0.2	16.37 (8.79,23.51)	2.3	87.20 (65.29,95.41)	1.4	71.39 (47.48,84.68)
Ahnuhati	2.6	90.21 (69.76,96.93)	0.7	46.51 (27.53,60.86)	0.2	16.37 (8.79,23.51)	1.9	81.70 (58.27,92.16)	0.3	23.52 (12.89,33.10)
Lull	2.6	90.21 (69.76,96.93)	0.7	46.51 (27.53,60.86)	0.2	16.37 (8.79,23.51)	1.9	81.70 (58.27,92.16)	0.3	23.52 (12.89,33.10)

Table 6.3: Mean abundances, P, of motile L. salmonis on juvenile pink salmon and estimated parasite induced host mortality, M, (with 95% credible intervals) for exposed populations during infestations.

Chapter 7

General Discussion

Humans have wrought widespread devastation of the Earth's ecosystems (Vitousek et al., 1997; Chapin et al., 2000). The global depletion of ocean ecosystems and the domestication and industrial production of marine species has occurred within the time span of a single human generation (Naylor et al., 2000; Myers and Worm, 2003; Duarte et al., 2007). Nearly half the seafood consumed by humans now comes from aquaculture as the global demand for seafood continues to grow faster than the supply from mostly fully exploited and overexploited wild fisheries (FAO, 2007). As fishing reduces wild fish abundances, parasite transmission declines (Anderson and May, 1978; Arneberg et al., 1998) and we see declines in the occurrence of disease outbreaks in wild fish (Ward and Lafferty, 2004) - a process known as *fishing out parasites* (Dobson and May, 1987). Yet at the same time, diseases have emerged spectacularly in farm fish and many diseases have emerged in wild fish in association with aquaculture (Heggberget et al., 1993; Gaughan, 2001; Murray and Peeler, 2005; Nowak, 2007). These trends suggest infectious diseases may undermine aquaculture's potential to augment global fish supply and restore wild stocks (Naylor et al., 2000). Because aquaculture primarily occurs in open net pens, farm fish are exposed to pathogens carried by wild fish and can then spread pathogens in new and damaging ways (Heggberget et al., 1993; Gaughan, 2001). Infectious diseases behave differently in the oceans than they do on land (McCallum et al., 2003), partly because the oceans are open systems in which pathogens are long-lived (McCallum et al., 2004). As aquaculture continues to grow, the sustainability of marine ecosystems and fisheries will depend, in part, on understanding marine epidemiology and implementing appropriate conservation policy (Bakke and Harris, 1998; Rosenberg, 2008).

Salmon lice are an emerging pathogen of farm salmon (Murray and Peeler, 2005; Nowak, 2007) and wild juvenile salmon sympatric with salmon farms (MacKenzie et al., 1998; Tully et al., 1999; Bjørn and Finstad, 2002; Morton et al., 2004). Farm

salmon first become infected with salmon lice that ultimately originate from wild salmon, and in Pacific Canada, this probably occurs when infected wild adult salmon pass farms on their spawning migration (Beamish et al., 2005). But the role of salmon farms in infestations of wild juvenile salmon have long been contentious (McVicar, 1997). Critics have argued that these linkages between salmon farms and salmon lice infestations of wild juvenile salmon are correlative and do not demonstrate causation (McVicar, 1997, 2004; Brooks, 2005). They point out that there are hosts for salmon lice other than salmon farms, and in particular, stickleback in the Broughton Archipelago have high salmon louse abundances which may be a source of lice infesting the wild juvenile salmon (Jones et al., 2006*b*). Critics such as Brooks (2005) highlight that abiotic factors known to affect salmon lice survival and development, such as temperature (Stien et al., 2005) and salinity (Bricknell et al., 2006), have natural variation that may underlie the infestations. Others cite experimental studies of louse infection challenges with juvenile pink and chum salmon showing high louse mortality and high salmon survival (Jones et al., 2006*a*, 2007; Webster et al., 2007). Although lice are pathogenic to juvenile salmon (Morton and Routledge, 2005), the impact of the infestations on wild salmon population dynamics have also been long contentious because there are many factors that affect salmon population dynamics and there have been no quantitative analyses testing for this effect (McVicar, 2004; Hilborn, 2006).

In this thesis, I have sought to advance our understanding of sea lice and salmon ecology by developing and applying a quantitative framework that combines mechanistic models with large data sets of sea lice transmission and wild salmon population dynamics. In chapter 2, I presented the field methods that were fundamental to subsequent chapters. In chapter 3, I combined large data sets and mathematical models of sea lice infecting wild juvenile salmon migrating past salmon farms. That analysis tested for and quantified the transmission of sea lice from farm salmon as well as naturally occurring hosts to wild juvenile salmon. Both farm salmon and natural hosts made significant contributions to sea lice infecting the wild juvenile salmon, but farm salmon were overwhelmingly the dominant source of lice. In chapter 4, I combined the transmission model developed in chapter 3 with large data sets and mathematical models of the survival of infected juvenile salmon. This analysis extensively replicated the results observed in chapter 3, spatially, temporally, and taxonomically and also estimated the wild juvenile salmon mortality caused by farm-origin lice. In chapter 5 I presented empirical and theoretical support for migratory allopatry in mediating pink salmon and sea lice population dynamics. Finally, in chapter 6, I tested for the effect of salmon lice infestations on wild pink salmon population dynamics. That analysis shows that pink salmon populations in

the Broughton Archipelago were rapidly declining during the salmon lice infestations between the years 2001-2005. The rate of decline estimated from analyzing annual pink salmon escapement estimates was very similar as that estimated from the survival of juvenile pink salmon held in ocean enclosures for 30 days. The analyses suggest salmon farm can undermine natural transmission barriers and have driven recurrent salmon lice infestations of wild juvenile pink and chum salmon leading to rapid declines and possible local extinction of at least pink salmon populations in the Broughton Archipelago.

But what of the critics? The stickleback hypothesis is easily rejected because early life stages of sea lice dominate infestations of juvenile wild salmon near salmon farms (Chapters 3-4; Morton and Williams, 2003; Morton et al., 2004; Morton et al. 2005; Morton et al. 2008), whereas lice on stickleback do not survive to reproductive age (Jones et al., 2006*b*,*a*). Also, Stickleback are widespread in British Columbia, whereas sea lice infestations on juvenile salmon are only known to have occurred near salmon farms (Chapters 3-4; Morton and Williams, 2003; Morton et al., 2004; Morton et al. 2005; Morton et al. 2008). There are natural hosts in the environment that I detected in the analyses in Chapters 3-4, and these hosts include local small populations of cutthroat trout (*O. clarki*) and dolly varden (*Salvelinus malma*) and scattered coastal ocean rearing Chinook (*O. tshawytscha*). The louse developmental rates I estimated at the ocean temperatures observed in Chapters 3 and 4 are related to those expected from experimental data (Stien et al., 2005) by the average juvenile salmon migration speed, $\sim$ 1 km per day. Salinity is vertically distributed in the water column and larval lice migrate vertically each day (Heuch, 1995) and can select favoured locations (Heuch, 1995). Louse behavior can combine with these physical variables - including tides, currents, and wind- to generate spatial distributions of nauplii and copepodids that take a variety of forms, including those observed in the Boughton Archipelago (Gillibrand and Kate, 2007). Brooks (2005) attempted to refute the link between farms and sea lice infestations of wild juvenile salmon in the Broughton Archipelago (Morton and Williams, 2003; Morton et al., 2004, 2005) based on the output of numerical oceanographic models that overestimate the dispersion of lice because they ignore louse behaviour and wind (Krkošek et al., 2006*b*). In Chapters 3-4, I fit models of sea lice dispersion from salmon farms that fully tracked sea lice development through nauplii and then copepodid stages, and in Chapter 4, they were further constrained by the current speeds measured in the Broughton Archipelago. This constrained model, which represents the simplest possible mathematical abstraction of sea lice dispersal, explains the data very well and has been spatially, temporally, and taxonomically replicated.

Laboratory experimental work by others (Jones et al., 2006*b*, 2007; Webster et al.,

2007) as well as field-based experiments I conducted in Chapter 4 all suggest high mortality of salmon lice on juvenile pink and chum salmon. This does not mean, however, that small juvenile pink and chum salmon are resistant to salmon lice. The work by Jones et al. (2006*b*, 2007) used juvenile salmon that weighed over 10 g and were fully scaled whereas the juvenile pink salmon being infested with salmon lice in the Boughton Archipelago weigh less than 1 g and do not have scales. It is well known that the effect of salmon lice on salmon survival is host size dependent (Pike and Wadsworth, 2000; Boxaspen, 2006), and so the studies by Jones et al. (2006*b*, 2007) probably overestimate the survival of juvenile pink salmon at the sizes they are exposed to salmon lice in the Boughton Archipelago. The study by Webster et al. (2007) was not designed to test for the effect of salmon lice on juvenile pink salmon survival. Rather, it was designed to test for the effect of salmon lice on the behavior of juvenile pink salmon and found that louse infections altered the salinity preference of juvenile salmon (Webster et al., 2007). In that study, the juvenile pink salmon that were examined after 14 days had lost most of their lice but those that remained infected had signs of pathology (Webster et al., 2007). It is the motile lice, which arise after 14 days of development, that are pathogenic to juvenile pink salmon (Chapter 4). Those studies that have raised lice to motile stages have observed high mortality of infected juvenile salmon (Figure 1.3 d; Chapter 4; Morton et al., 2005). In the field, the juvenile pink and chum salmon are chronically exposed to salmon lice copepodids, for the first 2-3 months of their marine life (Chapter 4). In this situation of chronic exposure, resistance to individual lice no longer protects the juvenile salmon. The high louse mortality may be compensated by the continued rate of new infections and so the high louse abundances and the epizootic mortality emerge.

The evidence that salmon farms caused the infestations and pink salmon population declines in the Broughton Archipelago is extensive. Where there are no salmon farms, louse abundance is low on pink salmon during early marine life because the vast majority of wild adult salmon that carry the parasite are offshore (Groot and Margolis, 1991; Chapter 5). Salmon farms produce large quantities of naupliar and copepodid lice during the early marine life of wild pink salmon in the Broughton Archipelago (Orr, 2007) and many studies have documented the transmission from farm to wild juvenile salmon (Chapters 3-4; Morton et al., 2003; Morton et al., 2004; Morton et al. 2005; Morton et al. 2008). I tested for the effect of salmon lice infestations on pink salmon population dynamics and controlled for other factors by using a comparative approach. Pink salmon population dynamics in areas exposed and unexposed to salmon farms were nearly identical before lice infestations began in the exposed area. During the infestations exposed populations

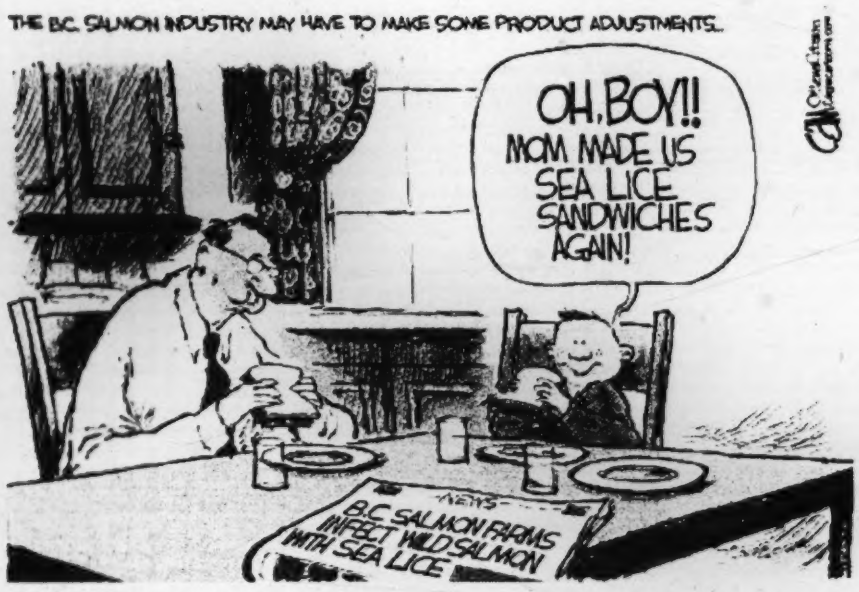
THE B.C. SALMON INDUSTRY MAY HAVE TO MAKE SOME PRODUCT ADJUSTMENTS.
OH, BOY!! MOM MADE US SEA LICE SANDWICHES AGAIN!
NEWS
B.C SALMON FARMS INFECT WILD SALMON WITH SEA LICE

Figure 7.1: A cartoon featuring sea lice appeared in the Ottawa Citizen shortly after the publication of Chapter 3 in Krkošek et al. (2005a).

declined significantly whereas unexposed populations remained productive. Because the two areas share the many factors that affect pink salmon population dynamics - evidenced by their synchronous fluctuations (Pyper et al., 2001) - but differ in lice infestations (Chapters 3-4; Morton et al., 2003; Morton et al., 2004; Morton et al. 2005), the infestations likely caused the difference in pink salmon population dynamics between the two areas. Lice are pathogenic to juvenile pink salmon during early marine life (Chapter 4; Morton and Routledge 2005) and the mortality rate of infected pink salmon is very similar when estimated from small scale experiments (Chapter 4) and analyzing multi-year multi-population escapement and infestation data (Chapter 6). The predicted mean motile *L. salmonis* abundance on juvenile pink salmon differentiating pink salmon population persistence and extinction was predicted to be 1-5 in Chapter 5 and estimated to be 1.35 in Chapter 6. These quantitative mechanistic linkages form a consilience of scientific evidence that indicates causal processes are underway, not spurious correlation.

7.1 Remarks on science in policy development

The results of this thesis have implications for policy. Infectious disease dynamics in coupled wild-farm fish systems see the intersection of many lines of human

and ecological concern. Wild Pacific salmon in particular, are fundamental to coastal ecosystems in the temperate Pacific northern hemisphere (Schindler et al., 2003). Coastal aboriginal peoples have a historical cultural legacy and contemporary subsistence dependency on wild Pacific salmon. Coastal economies contain a rapidly growing wilderness tourism sector whose economic viability is linked to wild salmon. Coastal economies also have strong dependency on fisheries for both subsistence and commercial exploitation. From a global food security perspective this thesis shows that the current model for aquaculture production - open net pens - can create new infectious disease dynamics that yield a net loss of food supply. These linkages have led to a wide array of science and policy initiatives involving regional and national governments, first nation's peoples, industry, environmental organizations, and government and academic scientists. Examples include symposia held by the National Organic Standards Board of the United States Department of Agriculture on net pen fish farming, the Special Committee on Sustainable Aquaculture of the British Columbia Legislative Assembly, the British Columbia Pacific Salmon Forum, and the World Wildlife Fund Salmon Aquaculture Dialogue. I have participated in these initiatives, and have also engaged in informing policy by communicating science to the public. Following the publication of chapters 3, 4, and 6 (Krkošek et al., 2005b, 2006*a*, 2007*a*), I worked hard to communicate their important scientific results by developing press and public outreach material as well as maintaining a web page that tracks public critiques of the work and my responses. These efforts have earned the coverage of over 500 news articles by, among others, *Science*, *Nature*, *The New York Times*, *The Economist*, and *The Globe and Mail*, as well as at least two political cartoons (Figures 7.1 and 7.2). Other highlights include interviews with Mary Lou Finlay on CBC Radio's *As It Happens* and John Nielson on NPR news, as well as other television and radio programs. These outreach and policy activities can be challenging as scientific information can be confused and misinterpreted by journalists, policy makers, and the public. In addition, I have witnessed special interest groups blatantly misrepresent scientific results as they try to influence policy and public opinion. Although taxing, these experiences have directly confirmed for me the important social responsibility of scientists to communicate effectively with policy makers and the public (Lubchenco, 1998).

7.2 Outstanding questions for future research

This thesis has made some quantitative advances in understanding sea lice and salmon population dynamics. There is much work to be done still to synthesize the models and data. The transmission model (Chapter 3), the survival model (Chapter 4), and the salmon population dynamics model (Chapters 5-6) are not

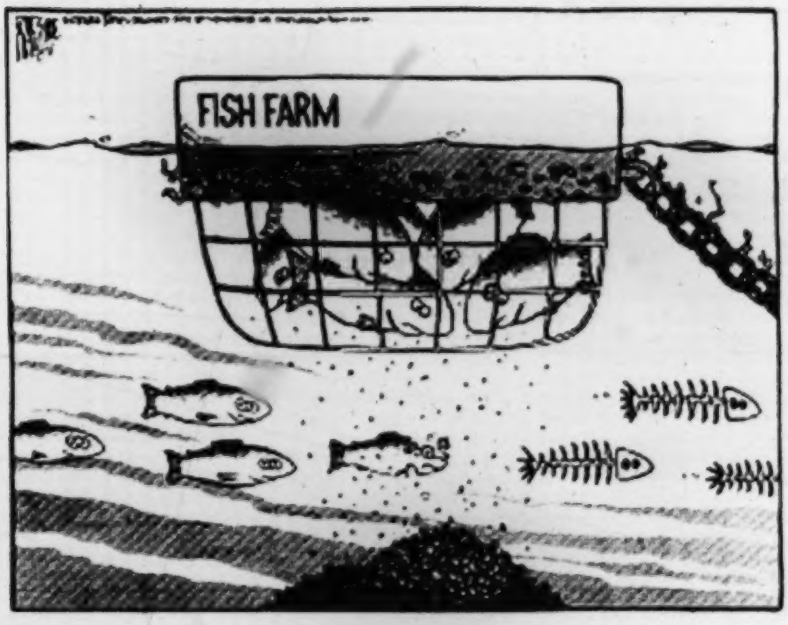

Figure 7.2: A cartoon featuring sea lice appeared in the Victoria Times Colonist shortly after the publication of Chapter 4 in Krkošek et al. (2006*a*).

fully linked. I have taken a first step towards this linkage in Chapter 5, where the juvenile salmon mortality due to sea lice in the Ricker model can be expressed as a within-year continuous-time survival function similar to White and Grenfell (1997). To achieve the synthesis, the transmission model needs to be extended, via a partial differential equation, to fully track the spatiotemporal dynamics of sea lice on migrating juvenile salmon rather than treating each dataset as a separate temporal snapshot of the spatial pattern of sea lice. This would allow one to extend also the dispersion submodel for nauplii and copepodids spreading from salmon farms and so accommodate temporal dynamics in the rate of nauplii release from farms, a scenario that may occur after farm salmon are chemically treated to remove sea lice. The infection-diffusion-decay equations describing the spread of nauplii and copepodids must then be numerically solved to generate the spatiotemporal distribution of copepodids through which the juvenile salmon migrate. Then by linking the solution for the sea lice dynamics on the juvenile salmon to the models of sea lice pathogenicity, the spatial data and models of sea lice on migrating juvenile salmon can supply the survival term in the Ricker model. This provides a prediction on the effects of sea lice on pink salmon population dynamics and can be compared with an independent analysis of pink salmon escapement data. Part of this synthesis will involve reconciling the value of $e^r = e^{1.2} = 3.3$ (Chapter 6) with the pink salmon

$R_0 \sim 17$ (Chapter 5) because these quantities should be equal; the conservative assumptions in chapter 5 (e.g. 100% fertilization success) need to be relaxed. While these are exciting theoretical ideas, the reality is that only a few pink salmon populations in the Broughton Archipelago can be represented this way because the spatial modeling represents fish from Glendale, Ahnuhati, and Lull systems, less so the Ahta and Kakweiken, and not at all the other rivers. There are few populations that can be tracked within the escapement data, a dataset that is famously noisy.

This thesis is a beginning for quantitatively understanding sea lice and salmon population dynamics with which to evaluate the possible management options to protect wild salmon. The management options include chemical treatment, moving/closing particular farms, reducing farm salmon density, and closed containment technology. By expanding the transmission model to include full spatiotemporal dynamics, including chemical treatment and sea lice dynamics on salmon farms, it is possible to measure the response in wild salmon infections and population dynamics. The modeling framework makes it possible also to quantitatively evaluate the various geometries in farm locations and how that effects wild salmon infections and population dynamics. Reducing farm salmon density is a management option based on the idea of thresholds in epidemiology (Lloyd-Smith et al., 2005). The most fundamental quantity in epidemiology is the net reproductive value, R_0 (Heesterbeek, 2002), which for sea lice determines the number of adult female lice produced by a single female louse in her lifetime. R_0 is often a function of host density, and can be used to identify the host density threshold differentiating disease eradication and epidemics (Grenfell and Dobson, 1995). I am not aware of a calculation for R_0 for sea lice, but it would be worthwhile to use it as a framework to analyze sea lice data on the salmon farms over the course of their industrial growth. The industry has grown in both the number of salmon farms and the number of fish per farm. It may be possible to identify a threshold farm salmon abundance below which sea lice outbreaks are suppressed. The management options impose costs on industry, and the largest probably come from closed containment technology because it requires large energy consumption to pump and treat material. The energy demands of closed containment technology may create other environmental costs such as carbon emission which may not be fully appreciated.

There are many questions in sea lice and salmon ecology that have emerged during my Ph.D. research but have not made it into this thesis. For example, juvenile salmon naturally experience very high mortality owing to predation and this may interact with parasitism to affect salmon population dynamics. Other studies have shown that predation can occur preferentially on infected prey (Mesa et al., 1998), and this can dampen predator-prey oscillations (Hudson et al., 1992). In chapter

5, I theoretically showed that compensatory predation can create a threshold louse abundance below which lice do not affect salmon population dynamics. External to this thesis, I have conducted some experiments and analysis in collaboration with Dr Larry Dill's lab investigating the effects of lice on juvenile salmon schooling behavior, predation risk and population dynamics that will address this issue and may have conservation implications. The outcome of this predation research should inform the modeling synthesis described above by explicitly including compensatory predation in the analysis of pink salmon escapement data. These predation experiments have led to another discovery that motile lice can escape predation on their host (Connors et al., 2008), a previously unknown but extraordinary behavior. There is also the need to reconcile the observations of high juvenile salmon resistance to sea lice from laboratory experiments (Jones et al., 2006*a*) as opposed to epizootic mortality in the field (Chapter 4). To address this I have expanded the McKendrick von Foerster equation (Kot, 2001) to model juvenile salmon and louse population dynamics in ocean enclosures following their collection in the field. This is a promising quantitative means by which to evaluate the difference between brief (experimental) and chronic (field) exposure to copepodids on sea lice population dynamics and juvenile salmon survival. There are also the effects of sea lice on juvenile salmon behavior (Webster et al., 2007) which if they extend to affect salmon migration behavior, for example by reducing migration speed, this may have important consequences for modeling and measuring sea lice transmission and juvenile salmon survival. Such questions go to the heart of juvenile salmon ecology, an area in which there is little known.

7.3 Concluding remarks

Aquaculture is important to future human food production (FAO, 2007). As the global demand for seafood continues to grow, so too will aquaculture because most wild fisheries are fully exploited or overexploited (Hilborn et al., 2003*a*). Already nearly half the seafood consumed by humanity comes from aquaculture rather than wild fisheries (FAO, 2007). This transition represents a fundamental and rapid change in how humans interact with ocean ecosystems (Duarte et al., 2007). The model for large scale industrial aquaculture production is based on open net pens, which can lead to pollution (Folke et al., 1994), invasive species (Volpe et al., 2001, 2000), genetic contamination of wild stocks (Fleming et al., 2000), and as I have shown here, new disease dynamics that can threaten wild stocks. These interactions, in varying degrees, underly global declines in wild salmon stocks sympatric with salmon farms (Ford and Myers, 2008). For salmon and sea lice, aquaculture transitions a native and normally benign host-parasite system into one

that threatens the host population and its highly valued ecosystem services. This happens because aquaculture increases the parasite exposure of juvenile salmon when they would normally be protected by *migratory allopatry* - a period of migration driven allopatry between uninfected juvenile and infected adult hosts. But aquaculture and fisheries are a complex adaptive human-environment system (Folke et al., 2004; Liu et al., 2007), in which politics, economics, ecology, and evolution are very much a part of the process. The Broughton salmon stocks probably will not go extinct because sea lice are not a problem without a solution, given sufficient political will (Rosenberg, 2008), but undoubtably there are also surprises and more challenges ahead. Disease control in aquaculture relies on chemicals and so the Red Queen dynamic emerges - humans continually invent new chemical defenses that are continually selecting for chemical resistance in pathogens. The oceans are open systems in which pathogens are long-lived (McCallum et al., 2003; Harvell et al., 2004; McCallum et al., 2004) and host species have highly dispersed or migratory lifecycles (Strathmann et al., 2002; Krkošek et al., 2007*b*). These properties mean that industrial net pen aquaculture can dramatically change disease dynamics, in ways that are damaging to the industry as well as ocean ecosystems and fisheries. As aquaculture grows, the epidemiology of wild-farm fish systems will likely feature prominently in the sustainability of coastal ecosystems and economies.

Literature cited

Anderson, R. and R. May. 1978. Regulation and stability of host-parasite population interactions. 1. Regulatory processes. Journal of Animal Ecology **47**:219–247.

Arneberg, P., A. Skorping, B. Grenfell, and A. Read. 1998. Host densities as determinants of abundance in parasite communities. Proceedings of the Royal Society of London Series B **265**:1283–1289.

Bakke, T. A. and P. D. Harris. 1998. Diseases and parasites in wild atlantic salmon (*Salmo salar*) populations. Canadian Journal of Fisheries and Aquatic Sciences **55**:247–266.

Beamish, R., S. Jones, C. Neville, R. Sweeting, G. Karreman, S. Saksida, and E. Gordon. 2006. Exceptional marine survival of pink salmon that entered the marine environment in 2003 suggests that farmed Atlantic salmon and Pacific salmon can coexist successfully in a marine ecosystem on the Pacific coast of Canada. ICES Journal of Marine Science **63**:1326–1337.

Beamish, R., C. Neville, R. Sweeting, and N. Ambers. 2005. Sea lice on adult Pacific salmon in the coastal waters of Central British Columbia, Canada. Fisheries Research **76**:198–208.

Bjørn, P. and B. Finstad. 2002. Salmon lice, *Lepeophtheirus salmonis* (Krøyer), infestation in sympatric populations of artic char, *Salvelinus alpinus* (L.), and sea trout, *Salmo trutta* (L.), in areas near and distant from salmon farms. ICES Journal of Marine Science **59**:131–139.

Blazer, V. 1992. Nutrition and disease resistance in fish. Annual Review of Fish Diseases **2**:309–323.

Boxaspen, K. 2006. A review of the biology and genetics of sea lice. ICES Journal of Marine Science **63**:1304–1316.

Bricknell, I. R., S. J. Dalesman, B. O'Shea, C. C. Pert, and A. J. M. Luntz. 2006. Effect of environmental salinity on sea lice *Lepeophtheirus salmonis* settlement success. Diseases of Aquatic Organisms **71**:201–212.

Brooks, K. 2005. The effects of water temperature, salinity, and currents on the survival and distribution of the infective copepodid stage of sea lice (*Lepeophtheirus salmonis*) originating on Atlantic salmon farms in the Broughton Archipelago of British Columbia, Canada. Reviews in Fisheries Science **13**:177–204.

Bruno, J. F., L. E. Petes, C. D. Harvell, and A. Hettinger. 2003. Nutrient enrichment can increase the severity of coral diseases. Ecology Letters **6**:1056–1061.

Bruno, J. F., E. R. Selig, K. S. Casey, C. A. Page, B. L. Willis, C. D. Harvell, H. Sweatman, and A. M. Melendy. 2007. Thermal stress and coral cover as drivers of coral disease outbreaks. PLoS Biology **5**:1220–1227.

Bunce, A. and F. I. Norman. 2000. Changes in the diet of the Australasian gannet (*Morus serrator*) in response to the 1998 mortality of pilchards (*Sardinops sagax*). Marine and Freshwater Research **51**:349–353.

Burnham, K. and D. Anderson. 2002. Model selection and multimodal inference: a practical information theoretic approach. Springer, New York.

Caswell, H. 2002. Marix population models. Sinauer, Sunderland.

Chapin, F. S., E. S. Zavaleta, V. T. Eviner, R. L. Naylor, P. M. Vitousek, H. L. Reynolds, D. U. Hooper, S. Lavorel, O. E. Sala, S. E. Hobbie, M. C. Mack, and S. Diaz. 2000. Consequences of changing biodiversity. Nature **405**:234–242.

Christie, K. S. and T. E. Reimchen. 2005. Post-reproductive pacific salmon, *Oncorhynchus* spp., as a major nutrient source for large aggregations of gulls, *Larus* spp. Canadian Field Naturalist **119**:202–207.

Connors, B., M. Krkošek, and L. Dill. 2008. Escape by sea lice from predation on their host. Biology Letters **in review**.

Costello, M. 2006. Ecology of sea lice parasitic on farmed and wild fish. Trends in Parasitology **22**:475–483.

Dann, P., F. I. Norman, J. M. Cullen, F. J. Neira, and A. Chiaradia. 2000. Mortality and breeding failure of little penguins, *Eudyptula minor*, in Victoria, 1995-96, following a widespread mortality of pilchard, *Sardinops sagax*. Marine and Freshwater Research **51**:355–362.

Darimont, C. T., T. E. Reimchen, and P. C. Paquet. 2003. Foraging behaviour by gray wolves on salmon streams in coastal british columbia. Canadian Journal of Zoology **81**:349–353.

Daszak, P., A. Cunninghamm, and A. Hyatt. 2000. Emerging infectious diseases of wildlife: threats to biodiversity and human health. Science **287**:443–449.

Dennis, B., P. Munholland, and J. M. Scott. 1991. Estimation of growth and extinction parameters for endangered species. Ecological Monographs **61**:115–143.

Dennis, B. and M. L. Taper. 1994. Density dependence in time series observations of natural populations: Estimation and testing. Ecological Monographs **64**:205–224.

Dobson, A. and R. May. 1987. The effects of parasites on fish populations - theoretical aspects. International Journal of Parasitology **17**:363–370.

Duarte, C., N. Marba, and M. Holmer. 2007. Rapid domestication of marine species. Science **316**:382–383.

Duffy-Anderson, J. T., K. Bailey, L. Ciannelli, P. Cury, A. Belgrano, and N. C. Stenseth. 2005. Phase transitions in marine fish recruitment processes. Ecological Complexity **2**:205–218.

Essington, T. E., A. H. Beaudreau, and J. Wiedenmann. 2006. Fishing through marine food webs. Proceedings of the National Academy of Sciences of the United States of America **103**:3171–3175.

Fallang, A., J. M. Ramsay, S. Sevatdal, J. F. Burka, P. Jewess, K. L. Hammell, and T. E. Horsberg. 2004. Evidence for occurrence of an organophosphate-resistant type of acetylcholinesterase in strains of sea lice (*Lepeophtheirus salmonis* Kroyer). Pest Management Science **60**:1163–1170.

FAO. 2007. State of the World's Fisheries and Aquaculture. Food and Agricultural Organization of the United Nations, Rome.

Fleming, I., K. Hindar, I. Mjolnerod, B. Jonsson, T. Balstad, and A. Lamberg. 2000. Lifetime success and interactions of farm salmon invading a native population. Proceedings of the Royal Society of London Series B **267**:1517–1523.

Folke, C., S. Carpenter, B. Walker, M. Scheffer, T. Elmqvist, L. Gunderson, and C. S. Holling. 2004. Regime shifts, resilience, and biodiversity in ecosystem management. Annual Review of Ecology Evolution and Systematics **35**:557–581.

Folke, C., N. Kautsky, and M. Troell. 1994. The costs of eutrophication from salmon farming - implications for policy. Journal of Environmental Management **40**:173–182.

Ford, J. and R. Myers. 2008. A global assessment of salmon aquaculture impacts on wild salmon. PLoS Biology **6**:e33.

Frank, K. T., B. Petrie, J. S. Choi, and W. C. Leggett. 2005. Trophic cascades in a formerly cod-dominated ecosystem. Science **308**:1621–1623.

Gao, F., E. Bailes, D. L. Robertson, Y. L. Chen, C. M. Rodenburg, S. F. Michael, L. B. Cummins, L. O. Arthur, M. Peeters, G. M. Shaw, P. M. Sharp, and B. H. Hahn. 1999. Origin of HIV-1 in the chimpanzee *Pan troglodytes troglodytes*. Nature **397**:436–441.

Gaughan, D. J. 2001. Disease-translocation across geographic boundaries must be recognized as a risk even in the absence of disease identification: the case with *Australian Sardinops*. Reviews in Fish Biology and Fisheries **11**:113–123.

Gaughan, D. J., R. W. Mitchell, and S. J. Blight. 2000. Impact of mortality, possibly due to herpesvirus, on pilchard *Sardinops sagax* stocks along the south coast of Western Australia in 1998-99. Marine and Freshwater Research **51**:601–612.

Gillibrand, P. A. and J. W. Kate. 2007. Dispersal of sea lice larvae from salmon farms: A model study of the influence of environmental conditions and larval behaviour. Aquatic Biology **1**:63–75.

Goldburg, R. and R. Naylor. 2005. Future seascapes, fishing, and fish farming. Frontiers in Ecology and the Environment **3**:21–28.

Grenfell, B. T. and A. P. Dobson. 1995. Ecology of Infectious Diseases in Natural Populations. Cambridge University Press, Cambridge.

Groot, C. and L. Margolis. 1991. Pacific salmon life histories. UBC Press, Vancouver, B.C.

Harvell, D., R. Aronson, N. Baron, J. Connell, A. Dobson, S. Ellner, L. Gerber, K. Kim, A. Kuris, H. McCallum, K. Lafferty, B. McKay, J. Porter, M. Pascual, G. Smith, K. Sutherland, and J. Ward. 2004. The rising tide of ocean diseases: unsolved problems and research priorities. Frontiers in Ecology and the Environment **2**:375–382.

Heard, W. 1991. Life history of pink salmon (*Oncorhynchus gorbuscha*). In: Pacific Salmon Life Histories (eds. Groot C and Margolis L). UBC Press, Vancouver, B.C.

Heesterbeek, J. A. P. 2002. A brief history of R_0 and a recipe for its calculation. Acta Biotheoretica **50**:189–204.

Heggberget, T. G., B. O. Johnsen, K. Hindar, B. Jonsson, L. P. Hansen, N. A. Hvidsten, and A. J. Jensen. 1993. Interactions between wild and cultured atlantic salmon - a review of the Norwegian experience. Fisheries Research **18**:123–146.

Heincke, F. 1908. Bericht ber die untersuchungen der biologischen anstalt auf helgoland zur naturgeschichte der nutzfische. Die Beteiligung Deutschlands an der Internationalen Meeresforschung **4/5**:67–155.

Heuch, P. A. 1995. Experimental-evidence for aggregation of salmon louse copepodids (*Lepeophtheirus salmonis*) in step salinity gradients. Journal of the Marine Biological Association of the United Kingdom **75**:927–939.

Heuch, P. A., P. A. Bjorn, B. Finstad, J. C. Holst, L. Asplin, and F. Nilsen. 2005. A review of the Norwegian 'national action plan against salmon lice on salmonids': The effect on wild salmonids. Aquaculture **246**:79–92.

Hilborn, R. 2006. Salmon-farming impacts on wild salmon. Proceedings of the National Academy of Sciences of the United States of America **103**:15277–15277.

Hilborn, R. 2007. Moving to sustainability by learning from successful fisheries. Ambio **36**:296–303.

Hilborn, R., T. A. Branch, B. Ernst, A. Magnussson, C. V. Minte-Vera, M. D. Scheuerell, and J. L. Valero. 2003*a*. State of the world's fisheries. Annual Review of Environment and Resources **28**:359–399.

Hilborn, R. and M. Mangel. 1997. The ecological detective, confronting models with data. Princeton University Press, Princeton.

Hilborn, R., T. P. Quinn, D. E. Schindler, and D. E. Rogers. 2003*b*. Biocomplexity and fisheries sustainability. Proceedings of the National Academy of Sciences of the United States of America **100**:6564–6568.

Hochachka, W. and A. Dhondt. 2000. Density-dependent decline of host abundance resulting from a new infectious disease. Proceedings of the National Academy of Sciences of the United States of America **97**:5303–5306.

Hocking, M. D. and T. E. Reimchen. 2006. Consumption and distribution of salmon (*Oncorhynchus* spp.) nutrients and energy by terrestrial flies. Canadian Journal of Fisheries and Aquatic Sciences **63**:2076–2086.

Holst, J. and A. McDonald. 2000. FISH-LIFT: a device for sampling live fish with trawls. Fisheries Research **48**:87–91.

Hudson, P., A. Dobson, and D. Newborn. 1992. Do parasites make prey vulnerable to predation? Red grouse and parasites. Journal of Animal Ecology **61**:681–692.

Hudson, P., A. Rizzoli, B. Grenfell, H. Heesterbeek, and A. Dobson. 2001. The Ecology of Wildlife Diseases. Oxford Univ Press, Oxford.

Hutchings, J. 2000. Collapse and recovery of marine fishes. Nature **406**:882–885.

Jackson, J., M. Kirby, W. Berger, K. Bjorndal, L. Botsford, B. Bourque, R. Bradbury, R. Cooke, J. Erlandson, J. Estes, T. Hughes, S. Kidwell, C. Lange, H. Lenihan, J. Pandolfi, C. Peterson, R. Steneck, M. Tegner, and R. Warner. 2001. Historical overfishing and the recent collapse of coastal ecosystems. Science **293**:629–638.

Johnsen, B. O. and A. J. Jensen. 1994. The spread of furunculosis in salmonids in Norwegian rivers. Journal of Fish Biology **45**:47–55.

Johnson, S. and L. Albright. 1991a. The development stages of *Lepeophtheirus salmonis*(Kroyer, 1837) (Copepoda: Caligidae). Canadian Journal of Zoology **69**:929–950.

Johnson, S. and L. Albright. 1991b. Development, growth, and survival of *Lepeophtheirus salmonis* (Copepoda, Caligidae) under laboratory conditions. Journal of the Marine Biological Association of the United Kingdom **71**:425–436.

Johnson, S. and M. Kent. 1992. Sea lice. In: Diseases in seawater netpet-reared salmonid fishes in the Pacific Northwest. (ed. Kent ML) Canadian Special Publication of Fisheries and Aquactic Sciences.

Johnson, S. C., J. W. Treasurer, S. Bravo, K. Nagasawa, and Z. Kabata. 2004. A review of the impact of parasitic copepods on marine aquaculture. Zoological Studies **43**:229–243.

Jolles, A., D. Cooper, and S. Levin. 2005. Hidden effects of chronic tuberculosis in African buffalo. Ecology **86**:2358–2364.

Jones, S., E. Kim, and S. Dawe. 2006*a*. Experimental infections with *Lepeophtheirus salmonis* (Kroyer) on threespine sticklebacks, *Gasterosteus aculeatus* L., and juvenile pacific salmon, *Oncorhynchus* spp. Journal of Fish Diseases **29**:489–495.

Jones, S. and A. Nemec. 2004. Pink Salmon Action Plan: Sea lice on juvenile salmon and on some nonsalmonid species caught in the Broughton Archipelago in 2003. Canadian Science Advisory Secretariat **Research Document**:www.dfo-mpo.gc.ca/csas/.

Jones, S., G. Prosperi-Porta, E. Kim, P. Callow, and N. Hargreaves. 2006*b*. The occurrence of *Lepeophtheirus salmonis* and *Caligus clemensi* (Copepoda : Caligidae) on three-spine stickleback *Gasterosteus aculeatus* in coastal British Columbia. Journal of Parasitology **92**:473–480.

Jones, S. R. M., M. D. Fast, S. C. Johnson, and D. B. Groman. 2007. Differential rejection of salmon lice by pink and chum salmon: disease consequences and expression of proinflammatory genes. Diseases of Aquatic Organisms **75**:229–238.

Kabata, Z. 1972. Developmental stages of *Caligus clemensi* (Copepoda: Caligidae). Journal of the Fisheries Research Board of Canada **29**:1571–1593.

Keeling, M. and B. Grenfell. 2002. Understanding the persistence of measles: reconciling theory, simulation and observation. Proceedings of the Royal Society of London Series B **269**:335–343.

Kent, M. L. 2000. Marine netpen farming leads to infections with some unusual parasites. International Journal for Parasitology **30**:321–326.

Kilpatrick, A., A. Chmura, D. Gibbons, R. Fleischer, P. Marra, and P. Daszak. 2006. Predicting the global spread of H5N1 avian influenza. Proceedings of the National Academy of Sciences of the United States of America **103**:19368–19373.

Kot, M. 2001. Elements of Mathematical Ecology. Cambridge University Press, Cambridge.

Krkošek, M., J. S. Ford, A. Morton, S. Lele, R. A. Myers, and M. A. Lewis. 2007*a*. Declining wild salmon populations in relation to parasites from farm salmon. Science **318**:1772–1775.

Krkošek, M., A. Gottesfeld, B. Proctor, D. Rolston, C. Carr-Harris, and M. A. Lewis. 2007*b*. Effects of host migration, diversity, and aquaculture on sea lice threats to pacific salmon populations. Proceedings of the Royal Society of London Series B **274**:3141–3149.

Krkošek, M., M. A. Lewis, A. Morton, L. N. Frazer, and J. P. Volpe. 2006*a*. Epizootics of wild fish induced by farm fish. Proceedings of the National Academy of Sciences of the United States of America **103**:15506–15510.

Krkošek, M., M. A. Lewis, and J. P. Volpe. 2005a. Transmission dynamics of parasitic sea lice from farm to wild salmon. Proceedings of the Royal Society of London Series B. **272**:689–696.

Krkošek, M., M. A. Lewis, J. P. Volpe, and A. Morton. 2006*b*. Fish farms and sea lice infestations of wild juvenile salmon in the Broughton Archipelago - a rebuttal to Brooks (2005). Reviews in Fisheries Science **14**:1–11.

Krkošek, M., A. Morton, and J. P. Volpe. 2005b. Non-lethal assessment of juvenile pink and chum salmon for parasitic sea lice infections and fish health. Transactions of the American Fisheries Society **134**:711–716.

Lafferty, K. D. 2004. Fishing for lobsters indirectly increases epidemics in sea urchins. Ecological Applications **14**:1566–1573.

Lafferty, K. D., J. W. Porter, and S. E. Ford. 2004. Are diseases increasing in the ocean? Annual Review of Ecology Evolution and Systematics **35**:31–54.

Liu, J. G., T. Dietz, S. R. Carpenter, M. Alberti, C. Folke, E. Moran, A. N. Pell, P. Deadman, T. Kratz, J. Lubchenco, E. Ostrom, Z. Ouyang, W. Provencher, C. L. Redman, S. H. Schneider, and W. W. Taylor. 2007. Complexity of coupled human and natural systems. Science **317**:1513–1516.

Lloyd, A. 2001. Destabilization of epidemic models with the inclusion of realistic distributions of infectious periods. Proceedings of the Royal Society of London Series B **268**:985–993.

Lloyd-Smith, J., P. Cross, C. Briggs, M. Daugherty, W. Getz, J. Latto, M. Sanchez, A. Smith, and A. Swei. 2005. Should we expect population thresholds for wildlife disease? Trends in Ecology and Evolution **20**:511–519.

Lotze, H. K., H. S. Lenihan, B. J. Bourque, R. H. Bradbury, R. G. Cooke, M. C. Kay, S. M. Kidwell, M. X. Kirby, C. H. Peterson, and J. B. C. Jackson. 2006. Depletion, degradation, and recovery potential of estuaries and coastal seas. Science **312**:1806–1809.

Lubchenco, J. 1998. Entering the century of the environment: A new social contract for science. Science **279**:491–497.

MacKenzie, K., M. Longshaw, G. S. Begg, and A. H. McVicar. 1998. Sea lice (*Copepoda: Caligidae*) on wild sea trout (*Salmo trutta L.*) in Scotland. ICES Journal of Marine Science **55**:151–162.

MacKinnon, B. 1998. Host factors important in sea lice infections. ICES Journal of Marine Science **55**:188–192.

Margolis, L. and Z. Kabata. 1988. Guide to the parasites of fishes of Canada. Part II - Crustacea. Canadian Special Publication of Fisheries and Aquactic Sciences.

Marshall, S. 2003. The incidence of sea lice infestations on wild sea trout compared to farmed salmon. Bulletin of the European Association of Fish Pathologists **23**:72–79.

McCallum, H. and A. Dobson. 1995. Detecting disease and parasite threats to endangered species and ecosystems. Trends in Ecology and Evolution **10**:190–194.

McCallum, H., D. Harvell, and A. Dobson. 2003. Rates of spread of marine pathogens. Ecology Letters **6**:1062–1067.

McCallum, H. I., A. Kuris, C. D. Harvell, K. D. Lafferty, G. W. Smith, and J. Porter. 2004. Does terrestrial epidemiology apply to marine systems? Trends in Ecology and Evolution **19**:585–591.

McVicar, A. 1997. Disease and parasite implications of the coexistence of wild and cultured Atlantic salmon populations. ICES Journal of Marine Science **54**:1093–1103.

McVicar, A. 2004. Management actions in relation to the controversy about salmon lice infections in fish farms as a hazard to wild salmonid populations. Aquaculture Research **35**:751–758.

Mesa, M., T. Poe, A. Maule, and C. Schreck. 1998. Vulnerability to predation and physiological stress responses in juvenile chinook salmon (*Oncorhynchus tshawytscha*) experimentally infected with *Renibacterium salmoninarum*. Canadian Journal of Fisheries and Aquatic Sciences **55**:1599–1606.

Morton, A. and R. Routledge. 2005. Mortality rates for juvenile pink (*Oncorhynchus gorbuscha*) and chum (O. keta) salmon infested with sea lice (*Lepeophtheirus salmonis*) in the Broughton Archipelago. Alaska Fisheries Research Bulletin **11**:146–152.

Morton, A., R. Routledge, C. Peet, and A. Ladwing. 2004. Sea lice (*Lepeophtheirus salmonis*) infection rates on juvenile pink (*Oncorhynchus gorbuscha*) and chum (*Oncorhynchus keta*) salmon in the near shore marine environment of British Columbia, Canada. Canadian Journal of Fisheries and Aquatic Sciences **61**:147–157.

Morton, A., R. Routledge, and R. Williams. 2005. Temporal patterns of sea lice infestation on wild Pacific salmon in relation to the fallowing of Atlantic salmon farms. North American Journal of Fisheries Management **25**:811–821.

Morton, A. and R. D. Routledge. 2006. Fulton's condition factor: Is it a valid measure of sea lice impact on juvenile salmon? North American Journal of Fisheries Management **26**:56–62.

Morton, A. and R. Williams. 2003. First report of the sea louse, *Lepeophtheirus salmonis*, infestation on juvenile pink salmon, *Oncorhynchus gorbuscha*, in nearshore habitat. Canadian Field Naturalist **117**:634–641.

Morton, A. M., R. Routledge, and M. Krkošek. 2008. Sea lice infestation of wild juvenile salmon and herring associated with fish farms off the east central coast of Vancouver Island, BC. North American Journal of Fisheries Management **in press**.

Murray, A. G. and E. J. Peeler. 2005. A framework for understanding the potential for emerging diseases in aquaculture. Preventive Veterinary Medicine **67**:223–235.

Myers, R., J. Baum, S. P. T.D. Shepherd, and C. Peterson. 2007. Cascading effects of the loss of apex predatory sharks from a coastal ocean. Science **315**:1846–1850.

Myers, R., K. Bowen, and N. Barrowman. 1999. Maximum reproductive rate of fish at low population sizes. Canadian Journal of Fisheries and Aquatic Sciences **56**:2404–2419.

Myers, R. and B. Worm. 2003. Rapid worldwide depletion of predatory fish communities. Nature **423**:280–283.

Nagasawa, K. 2001. Annual changes in the population size of the salmon louse *Lepeophtheirus salmonis* (Copepoda: Caligidae) on high-seas Pacific salmon (*Oncorhynchus* spp.), and relationship to host abundance. Hydrobiologia **453**:411–416.

Nash, R., A. Valencia, and A. Geffen. 2006. The origin of Fulton's condition factor - setting the record straight. Fisheries **31**:236–238.

Naylor, R., R. Goldburg, J. Primavera, N. Kautsky, M. Beveridge, J. Clay, C. Folke, J. Lubchenco, H. Mooney, and M. Troell. 2000. Effect of aquaculture on world fish supplies. Nature **405**:1017–1024.

Noakes, D. J., R. J. Beamish, and M. L. Kent. 2000. On the decline of Pacific salmon and speculative links to salmon farming in British Columbia. Aquaculture **183**:363–386.

Nowak, B. F. 2007. Parasitic diseases in marine cage culture - an example of experimental evolution of parasites? International Journal for Parasitology **37**:581–588.

Nylund, A., H. Plarre, M. Karlsen, F. Fridell, K. F. Ottem, A. Bratland, and P. A. Saether. 2007. Transmission of infectious salmon anaemia virus (ISAV) in farmed populations of Atlantic salmon (*Salmo salar*). Archives of Virology **152**:151–179.

Olivier, G. 2002. Disease interactions between wild and cultured fish - Perspectives from the American Northeast (Atlantic Provinces). Bulletin of the European Association of Fish Pathologists **22**:103–109.

Orr, C. 2007. Estimated sea louse egg production from Marine Harvest Canada farmed Atlantic salmon in the Broughton Archipelago, British Columbia, 2003-2004. North American Journal of Fisheries Management **27**:187–197.

Osterhaus, A. and E. J. Vedder. 1988. Identification of virus causing recent seal deaths. Nature **335**:20–20.

Paine, R. T., M. J. Tegner, and E. A. Johnson. 1998. Compounded perturbations yield ecological surprises. Ecosystems **1**:535–545.

Papoulis, A. 1963. Probability, random variables, and stochastic processes. McGraw-Hill, USA.

Parker, R. R. 1968. Marine mortality schedules of juvenile pink salmon of the Bella Coola River, central British Columbia. Journal of the Fisheries Research Board of Canada **25**:757–794.

Parker, R. R. and L. Margolis. 1964. A new species of parasitic copepod, *Caligus clemensi* sp. nov. (Caligoida: Caligidae), from pelagic fishes in the coastal waters of British Columbia. Journal of the Fisheries Research Board of Canada **21**:873–889.

Pauly, D., J. A. amd E. Bennett, V. Christensen, P. Tyedmers, and R. Watson. 2003. The future for fisheries. Science **302**:1359–1361.

Pauly, D., V. Christensen, J. Dalsgaard, R. Froese, and F. Torres. 1998. Fishing down marine food webs. Science **279**:860–863.

Peet, C. 2007. M.Sc. Thesis. Interactions between sea lice (*Lepeoptheirus salmonis* and *Caligus clemensii*) and juvenile chum (*Oncorhynchus keta*) and pink salmon (Oncorhynchus gorbusha) in British Columbia. University of Victoria .

PFRCC. 2002. Pacific Fisheries Resource Conservation Council 2002 Advisory: the protection of Broughton Archipelago pink stocks. In: Report to Minister of Fisheries and Oceans, and B.C. Minister of Agriculture, Food, and Fisheries. Pacific Fisheries Resource Conservation Council, Vancouver.

Pike, A. and S. Wadsworth. 2000. Sealice on salmonids: their biology and control. Advances in Parasitology **44**:233–337.

Pinheiro, J. and D. Bates. 2004. Mixed Effects Models in S and S-plus. Springer, New York.

Pollard, W. R., G. F. Hartman, C. Groot, and P. Edgell. 1997. Field identification of coastal juvenile salmonids. Harbour Publishing, Madeira Park, B.C.

Pulliam, H. R. 1988. Sources, sinks, and population regulation. American Naturalist **132**:652–661.

Pyper, B., F. Mueter, R. Peterman, D. J. Blackbourn, and C. Wood. 2001. Spatial covariation in survival rates of Northeast Pacific pink salmon (*Oncorhynchus gorbuscha*). Canadian Journal of Fisheries and Aquatic Sciences **58**:1501–1515.

Quinn, T. 2004. The Behavior and Ecology of Pacific Salmon and Trout. University of Washington Press, Seattle.

Reimchen, T. E. 2000. Some ecological and evolutionary aspects of bear-salmon interactions in coastal british columbia. Canadian Journal of Zoology **78**:448–457.

Revie, C. W., G. Gettinby, J. W. Treasurer, A. N. Grant, and S. W. J. Reid. 2002. Sea lice infestations on farmed Atlantic salmon in Scotland and the use of ectoparasitic treatments. Veterinary Record **151**:753–757.

Ricker, W. E. 1954. Stock and recruitment. Journal of the Fisheries Research Board of Canada **11**:559–623.

Ritchie, G. 1997. The host transfer ability of *Lepeophtheirus salmonis* (Copepoda: Caligidae) from farmed Atlantic salmon, *Salmo salar* L. Journal of Fish Diseases **20**:153–157.

Roberts, R. and C. Shepherd. 1997. Handbook of Salmon and Trout Diseases. Blackwell Science, Oxford.

Rohde, K. 2005. Marine Parasitology. CSIRO, Victoria.

Rosenberg, A. A. 2008. Aquaculture: The price of lice. Nature **451**:23–24.

Saksida, S. M. 2006. Infectious haematopoietic necrosis epidemic (2001 to 2003) in farmed atlantic salmon *Salmo salar* in British Columbia. Diseases of Aquatic Organisms **72**:213–223.

Salo, E. 1991. Life history of chum salmon (*Oncorhynchus keta*). In: Pacific Salmon Life Histories (eds. Groot C and Margolis L). UBC Press, Vancouver, B.C.

Schindler, D. E., M. D. Scheuerell, J. W. Moore, S. M. Gende, T. B. Francis, and W. J. Palen. 2003. Pacific salmon and the ecology of coastal ecosystems. Frontiers in Ecology and the Environment **1**:31–37.

Shaw, D. and A. Dobson. 1995. Patterns of macroparasite abundance and aggregation in wildlife populations: A quantitative review. Parasitology **111**:111–133.

Shaw, D., B. Grenfell, and A. Dobson. 1998. Patterns of macroparasite aggregation in wildlife host populations. Parasitology **111**:597–610.

Stien, A., P. Bjorn, P. Heuch, and D. Elston. 2005. Population dynamics of salmon lice *Lepeophtheirus salmonis* on Atlantic salmon and sea trout. Marine Ecology Progress Series **290**:263–275.

Strathmann, R. R., T. R. Hughes, A. M. Kuris, K. C. Lindeman, S. G. Morgan, J. M. Pandolfi, and R. R. Warner. 2002. Evolution of local recruitment and its consequences for marine populations. Bulletin of Marine Science **70**:377–396.

Tompkins, D. M. and M. Begon. 1999. Parasites can regulate wildlife populations. Parasitology Today **15**:311–313.

Tully, O., P. Gargan, W. R. Poole, and K. Whelan. 1999. Spatial and temporal variation in the infestation of sea trout (*Salmo trutta* L.) by the Caligid copepod *Lepeophtheirus salmonis* (Kroyer) in relation to sources of infection in Ireland. Parasitology **119**:41–51.

Vitousek, P. M., H. A. Mooney, J. Lubchenco, and J. M. Melillo. 1997. Human domination of earth's ecosystems. Science **277**:494–499.

Volpe, J., B. Anholt, and B. Glickman. 2001. Competition among juvenile Atlantic salmon (*Salmo salar*) and steelhead (*Oncorhynchus mykiss*): relevance to invasion potential in British Columbia. Canadian Journal of Fisheries and Aquatic Sciences **58**:197–207.

Volpe, J., E. Taylor, D. Rimmer, and B. Glickman. 2000. Evidence of natural reproduction of aquaculture-escaped Atlantic salmon in a coastal British Columbia river. Conservation Biology **14**:899–933.

Ward, J. R. and K. D. Lafferty. 2004. The elusive baseline of marine disease: Are diseases in ocean ecosystems increasing? PLoS Biology **2**:542–547.

Ward, T. M., F. Hoedt, L. McLeay, W. F. Dimmlich, G. Jackson, P. J. Rogers, and K. Jones. 2001*a*. Have recent mass mortalities of the sardine *Sardinops*

sagax facilitated an expansion in the distribution and abundance of the anchovy *Engraulis australis* in South Australia? Marine Ecology Progress Series **220**:241–251.

Ward, T. M., F. Hoedt, L. McLeay, W. F. Dimmlich, M. Kinloch, G. Jackson, R. McGarvey, P. J. Rogers, and K. Jones. 2001*b*. Effects of the 1995 and 1998 mass mortality events on the spawning biomass of sardine, *Sardinops sagax*, in South Australian waters. ICES Journal of Marine Science **58**:865–875.

Webster, S. J., L. M. Dill, and K. Butterworth. 2007. The effect of sea lice infestation on the salinity preference and energetic expenditure of juvenile pink salmon (*Oncorhynchus gorbuscha*). Canadian Journal of Fisheries and Aquatic Sciences **64**:672–680.

White, K. A. J. and B. T. Grenfell. 1997. Regulation of complex host dynamics by a macroparasite. Journal of Theoretical Biology **186**:81–91.

Wilkinson, C. E., M. D. Hocking, and T. E. Reimchen. 2005. Uptake of salmon-derived nitrogen by mosses and liverworts in coastal British Columbia. Oikos **108**:85–98.

Wonham, M., T. de Camino-Beck, and M. A. Lewis. 2004. An epidemiological model for West Nile Virus: Invasion analysis and control applications. Proceedings of the Royal Society of London Series B **271**:501–507.

Worm, B., E. Barbier, N. Beaumont, J. Duffy, C. Folke, B. Halpern, J. Jackson, H. Lotze, F. Micheli, S. Palumbi, E. Sala, K. Selkoe, J. Stachowicz, and R. Watson. 2006. Impacts of biodiversity loss on ocean ecosystem services. Science **314**:787–790.